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Sahni, B. (1936a) Wegener's theory of continental drift in the light of Palaeobotanical evidence. *J. Indian bot. Soc.*, 15: 31-32.

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CONTENTS

POECILOCERUS PICTUS FABR .. A PAINTED GRASSHOPPER PEST ON *CALOTROPIS GIGANTEA* IN SOUTH INDIA

By S. Venugopal .. 1

PHYSIOLOGY OF DISEASE IN PLANTS

By C. V. Subramanian .. 9

A NOTE ON *EUPHORBIA LONGISTYLA* BOISS

By K. Subramanyam & N. P. Balakrishnan .. 45

HOLOTRICHOUS CILIATES FROM MADRAS

By Anna J. Kottil .. 49

A CASE FOR SEED CERTIFICATION AND FOR ORGANISING CROP IMPROVEMENT ASSOCIATIONS IN INDIA

By T. Venkataramana Reddy .. 57

RECENT ADVANCES IN SORGHUM IMPROVEMENT

By T. Venkataramana Reddy .. 69

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J. Madras Univ., B. XXIX, No. 1, pages 1-8, 1959

Poecilocerus Pictus Fabr. . A Painted Grasshopper Pest on *Calotropis gigantea* in South India *

BY

S. VENUGOPAL, M.Sc.,

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(Received for publication, 16th July 1958)

Introduction: *Calotropis gigantea* a common shrub has been widely used as a green manure for paddy from very early times. Like other crops, it is subject to the attack of number of pests, viz., *Danais chrysippus W., Poecilocerus pictus Fb., Dacus longistylus W., Eurybrachys tomentosa T.,* and *Aphis neri B.* (Ramakrishna Iyer 1940).

Occurrence and Habit: The grasshopper *Poecilocerus pictus* fabr., (Acridiidae—Orthoptera) is the most important pest and has a fairly wide distribution throughout the plains of India except in Bihar, Bengal and Assam and in Africa. Kirby (1914) recorded the pest in Baluchistan, Quetta, Sind and Karachi. In Northern India, this pest has been observed to feed on a wild plant called 'ak' a species of the genus *Calotropis* and hence the name 'ak' grasshopper. Appearing in large numbers twice during the year, gregarious in habit and crowding together all over the main stem and lateral branches the pest has been observed to be feeding voraciously on the leaves (Plate I, Figs. 1, 2).

Alternate Host Plants: The pest has been observed to feed on fig (Fletcher, 1914), Citrus (Pruthi, 1954), and on Papaya, Bhendai, Canna, Luffa sp. and *Clerodendron* (Batra 1955). In Coimbatore, it was observed to feed on castor, cowpea, brinjal, bhendai, and cotton. It appears to have a preference to calotropis and eats up every part of it namely leaves, stem, pods and flowers and reduces the plant to nacked stems.

Life History: An attempt has been made by Pruthi and Nigam (1929) to work out the bionomics, life-history and control

* Formed part of M.Sc., thesis submitted to the Madras University in August, 1956.

of this grass-hopper. Asana and Makina (1934) have thrown some light on its chromosomes. The present study gives a comprehensive idea on its life history, bionomics and the latest control methods.

Copulation: It has been observed during two years of the study that the male grasshopper does not immediately start copulation, that it takes 6 to 9 days as far as pre-copulation period is concerned, and that copulation between male and female lasts for 5 to 7 hours. There is intermittant copulation between male and female within 22 to 26 days.

Oviposition: (Text-figs. 1, 2). The egg laying takes place after 25 to 30 days. The eggs are laid 6″ to 7″ below the soil surface (Plate I, Fig. 14). The nature of soil preferred is of loose texture. During the process of egg laying, the female thrusts its abdomen on the ground locating best spot for oviposition and constructs an egg-chamber with its ovipositor. The eggs are laid one by one in a spiral manner forming a compact mass. (Plate I, Fig. 3-5. After all the eggs are laid, the female slowly draws the abdomen and the vacant space is filled with a frothy mass at the top. The total number of eggs noted in an egg mass comes to 145 to 170.

Egg: It is rather elongated, slightly curved, thicker in the mid portion and tapering towards both extremities. The colour of the egg is orange. The egg period during autumn lasts for one month. The newly hatched nymphs always try to move upwards and catch at any piece of twig. The nymphs begin to feed one or two days after emergence, and each one undergoes six months before it becomes an adult. The total nymphal period roughly takes about 55 to 60 days during autumn (Table 1). In the case of nymphs hatching in summer, the nymphal period is very long. There is a tendency for majority of the eggs laid in summer to over winter for nearly 4 months and become adults in the following July after a nymphal period lasting from 73 to 77 days (Table 2).

First Nymphal Stage (Text-figs. 3, 4): Newly emerged nymphs are light orange in colour with prominent blackish and white stripes. They measure 8 to 11 mm. in length and 2.2 mm. in breadth round about thoracic region. The head is conical and jets out in front between the compound eyes. The eyes are oval and black. The antennnae are bright purple and eight segmented. The basal joint is cylindrical and the second is small and globular, the third

TABLE 1

Statement showing the summary of the life history of *Poecilocerus pictus*, Fabr. during autumn—1953-1955

Serial No.	Date of hatching	Date of 1st instar	Period in days	Date of 2nd instar	Period in days	Date of 3rd instar	Period in days	Date of 4th instar	Period in days	Date of 5th instar	Period in days	Date of 6th instar	Period in days	Total period in days
1.	2-9-53	12-9-53	10	20 -9-53	8	29 -9-53	9	7-10-53	9	16-10-53	9	26-10-53	10	55
2.	4-9-53	13-9-53	9	22 -9-53	8	30 -9-53	8	9-10-53	9	18-10-53	10	29-10-53	11	55
3.	15-9-53	26-9-53	12	5-10-53	9	13-10-53	8	22-10-53	9	2-11-53	11	13-11-53	11	60
4.	18-9-53	27-9-53	10	5-10-53	8	13-10-53	8	22-10-53	9	31-10-53	9	11-11-53	11	55
5.	6-9-54	15-9-54	10	23 -9-54	8	1-10-54	8	10-10-54	9	19-10-54	9	30-10-54	11	55
6.	8-9-54	19-9-54	12	21 -9-54	9	6-10-54	8	15-10-54	9	26-10-54	11	6-11-54	11	60
7.	10-9-54	23-9-54	10	1-10-54	8	5-10-54	8	14-10-54	9	22-10-54	9	3-11-54	11	55
8.	14-9-54	23-9-54	10	1-10-54	8	9-10-54	8	18-10-54	9	27-10-54	9	7-11-54	11	55
9.	8-9-55	19-9-55	12	28 -9-55	9	6-10-55	8	15-10-55	9	26-10-55	11	5-11-55	11	60
10.	12-9-55	21-9-55	10	29 -9-55	8	7-10-55	8	16-10-55	9	25-10-55	9	5-11-55	11	55
11.	14-9-55	25-9-55	12	4-10-55	8	12-10-55	8	20-10-55	9	9-10-55	11	12-11-55	11	60
12.	16-9-55	25-9-55	10	3-10-55	8	11-10-55	8	20-10-55	9	29-10-55	9	9-11-55	11	55
	Maximum		12		9		9		9		11		11	60
	Minimum		10		8		8		9		9		10	55
	Average		10·5		8·3		8-1		9		9·6		10·9	56·9

TABLE 2

Statement showing the summary of the life history of *Poecilocerus pictus*, Fabr. during Summer 1953-1955.

Serial No.	Date of hatching	Date of 1st instar	Period in days	Date of 2nd instar	Period in days	Date of 3rd instar	Period in days	Date of 4th instar	Period in days	Date of 5th instar	Period in days	Date of 6th instar	Period in days	Total period in days
1.	2-4-53	14-4-53	12	26-4-53	12	8-5-53	13	21-5-53	12	2-6-53	14	16-6-53	14	77
2.	8-4-53	20-4-53	12	3-5-53	13	15-5-53	12	28-5-53	13	9-6-53	12	22-6-53	13	75
3.	18-4-53	30-4-53	13	14-5-53	14	26-5-53	12	6-6-53	11	17-6-53	11	30-6-53	13	74
4.	20-4-53	3-5-53	13	12-5-53	12	25-5-53	13	7-6-53	12	20-6-53	13	3-7-53	13	76
5.	28-3-54	8-4-54	12	21-4-54	13	3-8-54	12	15-5-54	12	28-5-54	13	11-6-54	14	76
6.	4-4-54	16-4-54	12	29-4-54	13	11-5-54	12	25-5-54	13	6-6-54	12	20-6-54	14	76
7.	19-4-54	2-5-54	13	16-5-54	14	27-5-54		7-6-54	11	19-6-54	11	2-7-54	13	74
8.	25-4-54	7-5-54	13	19-5-54	12	31-5-54	13	13-6-54	12	25-6-54	13	6-7-54	12	75
9.	1-4-55	14-4-55	13	26-4-55	12	9-5-55	13	21-5-55	12	11-6-55	11	13-6-55	13	74
10.	4-4-55	17-4-55	13	30-4-55	12	12-5-55	13	25-5-55	12	6-6-55	12	18-6-55	13	75
11.	8-4-55	20-4-55	12	3-5-55	13	15-5-55	12	26-5-55	11	8-6-55	13	20-6-55	12	73
12.	10-4-55	23-4-55	13	5-5-55	12	18-5-55	13	30-5-55	12	13-6-55	13	25-6-55	12	75
	Maximum		13		13		13		12		13		14	77
	Minimum		12		12		12		11		11		12	73
	Average		12·5		12·5		12·5		11·9		12·2		12·9	75·7

to seventh segment being just equal. The eighth segment is very long and is equal in length when compared with the three preceeding segments. The first and second pairs of legs are small, transverse and with black and white markings all over. The third pair is stout. The upper and lower ridges of the hind femora have eight to nine and five to six black dots. The hind tarsi are three-jointed, the terminal joint being provided with a pair of claws and a median pad. Ten terga are visible in both sexes, and the last two being fused partially. In the male, there are ten sterna including the first one which is fused with the sternum of the metathorax. The ninth sternum is big with a ventral depression and deeply notched at the posterior margin. This represents the rudiment of sub-genital plate. Above this plate, there is a pair of triangular outgrowths known as podical plates. In the female also, ten sterna are clear, the ninth being cleft into two triangular pieces which are the rudiments of the dorsal valves of the ovipositor. At the posterior margin of the eighth sternum there is a very tiny pair of protuberances which are the rudiments of the ventral valves of the ovipositor.

Second Nymphal Stage (Text-figs. 3, 4): There is not much of colour variation in the second stage and it has a striking resemblance to first stage nymph. It measures 10 to 12 mm. in length and 2.4 mm. in breadth of the body. The antennnae measure 2.8 mm. in length and nine jointed. On the distal margin of the third to sixth joints, there are two small white spots which are unrepresented in the first stage. The sub-genital plate in the male is more pronounced. The ventral and dorsal valves of the ovipositor in the case of female has grown longer partially hiding the ninth segment and podical plates in the anterior half.

Third Nymphal Stage (Plate I, Fig. 11: On the dorsal aspect, there is a colour change from yellow to orange as far as median spots are concerned. The length of the body is 16 to 18 mm. The length of the antennae is about 4·2 mm. and eleven joined. The meso and meta thorax develop prominent triangular outgrowths which represent the rudiments of future wings. In the female the eighth segment has developed an inwardly directed cresentic fold near the base of the ventral valves. This fold ultimately becomes the egg guide clearly noted in the fifth nymphal stage.

Fourth Nymphal Stage (Plate I, Fig. 10): The body measures 19 to 23 mm. in length and 3 to 4·2 mm. in breadth. The eyes are brown and the transverse stripe has become broader. The anten-

nae are 5·5 mm. in length and 12 to 14 jointed. The postero-lateral angles of the terminal somites have become dark and develop oblique folds which form the inner pair of the valves of the ovipositor.

TABLE 3

Control trials on *Poecilocerus pictus* Fabr.

S. No.	Treatments	Initial counts	Counts after 24 hrs.	Counts after 48 hours	% of reduction in population after 48 hours
1.	B.H.C. 5%	25	14	4	84
2.	D.D.T. 5%	25	25	25	Nil
3.	B.H.C. 10%	25	5	Nil	Nil
4.	D.D.T. 10%	25	20	15	40
5.	Aldrin 2½%	25	4	Nil	52
6.	Dieldrin 2½%	25	4	Nil	100
7.	Control	25	25	25	..

TABLE 4

Statistical analysis of the control trials on *Poecilocerus, pictus,* Fabr.

S. No.	Treatments	Difference in proportion	Standard error	Significance
1.	D.D.T. 10% B.H.C. 10%	0·60	·13	Yes
2.	D.D.T. 10% Dieldrin 2½%	0·60	·13	Yes

Fifth Nymphal Stage (Plate I, Fig. 6): There is a distinct colour change to deep yellow in the place of white and yellow patches have also appeared. The nymphs have grown in size and measure 35 to 40 mm. in length and 6 to 7 mm. in breadth. The antennae measure about 7·8 mm. and consists of fifteen joints. In the case of female, the ventrals have grown much thicker and extend backwards almost reaching the dorsal valves and the egg guide has become very distinct.

Sixth Nymphal Stage (Plate I, Fig. 7): The freshly emerged nymph has a bright yellow colour with black markings replacing yellow patches of the previous stage. It measures about

45-50 mm. in length and 7·5 to 8 mm. in breadth. The eyes have become bluish in hue without any transverse strips. The antennae are fifteen jointed and measure 11 to 13·6 mm. The subgenital plate is conical inside which the aedeagus is situated. The egg guide is well developed and remains deep in the interior of the two ventral valves. The dorsal valves jet out far beyond podical plates by growing in thickness. There is bluish colour in the body and antennal region develops orange and yellow spots. The ventral and dorsal valves become chitinous.

The Final Moult: It is a pleasure to witness the final moult of the hopper. It stops feeding and takes rest on the twig. There is a forward and backward movement of the head and the pronotum is moved up and down which results in the rupture along the median line. This rupture gardually increases and goes as far as the wing pads. The head and thoracic region are gradually pushed out of their sacs. It is followed by the appearance of first and second pair of legs. Finally the cast skin is left behind as the adult finally emerges. After half an hour the fore-wings are fully spread out and develop bluish colour with light yellow veins.

Adult: A close description is given by Kirby (1914). Plate I, Figs. 12, 13).

Control Measures: Following stomach poisons were tried. Sodium arsenate as spray in the proportion of 2, 1 or ½ lb. and calcium arsenate as spray in 1 or 2 lb. in 100 gallons of water. Paris green diluted with chalk was dusted in the proportion of 8:1 and 6:1. These stomach poisons did not effect complete control of the hopper. Seven healthy calotropis plants were selected and caged and 25 adult grasshoppers were introduced in each. These plants were treated with BHC (5 and 10%), DDT 5 and 10%, Aldrin 2%, Dieldrin 2½% respectively and the seventh plant as control. Mortality counts were taken after 24 and 48 hours after treatment. These results have been presented in Table 3.

It will be observed from the table that BHC 5% and Dieldrin 2½% gave cent per cent kill. Next in descending order come BHC 5%, Aldrin 2½% and DDT 5%. DDT 5% did not effect any kill at all. In the controls of course, the insects remained quite healthy. These results were subjected to statistical analysis as shown in Table 4. It may be seen from the table that in between the different treatments BHC 10% and Dieldrin 2½% were highly significant and far superior to DDT 10%

SUMMARY

Poecilocerus pictus Fb. is the conspicuous painted grasshopper very commonly noted feeding on every part of the calotropis plant. It has a partiality for this plant and when the plant is not available, it proves to be a potential pest on brinjal, castor, tomato and citrus. There are at least two broods in a year. The nymph is coloured yellow with black stripples and red spots, the colouring gradually giving place to that of the adult in the last two instars before the final moult. The various instars have been described.

Many stomach poisons were tried as spray and dust. Among stomach poisons sodium arsenite at the dosage of ½ lb. per 100 gallons of water proved to be a satisfactory method of control. It is further stated here that BHC 10%, Dieldrin 2½% and Aldrin 2½% dust completely exterminated the grasshoppers. Hence *Poecilocerus pictus* flb. should be reckoned as a major pest on *Calotropis gigantea.*

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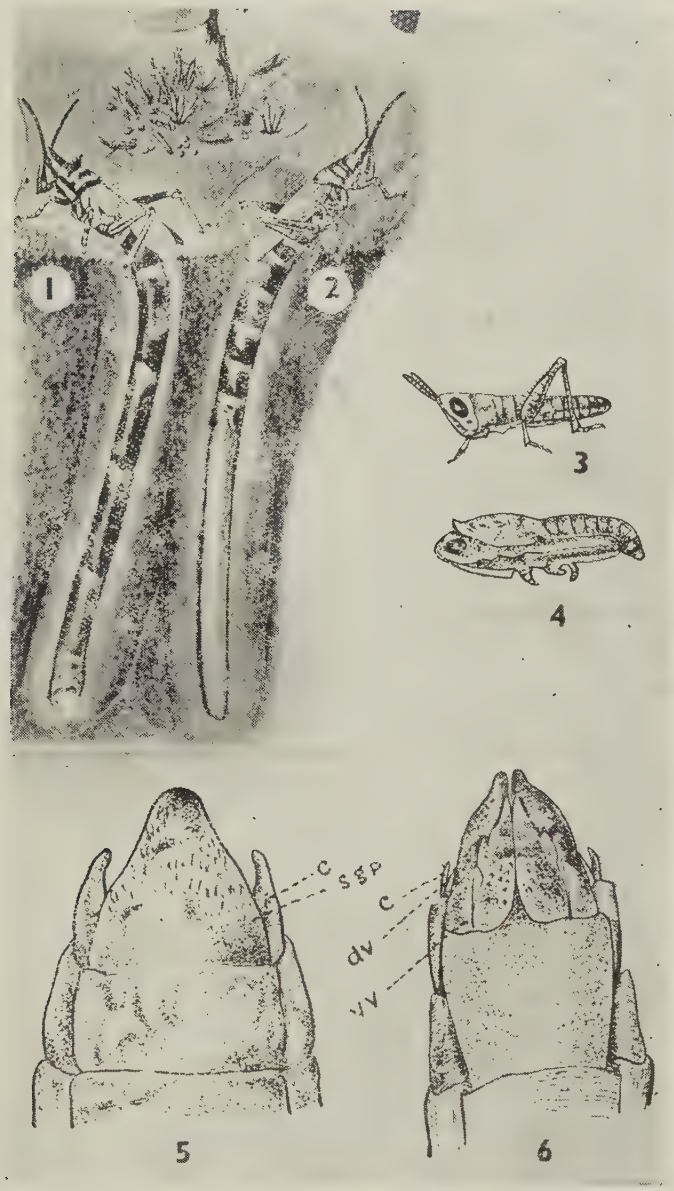
S. VENUGOPAL

FIGS. 1-2. indicates the gregarious habit of *Poecilocerus pictus* Fabr. nymphs clinging on to the naked stems devoid of any green matter. FIGS. 3, 4, 5 & 9. Eggs and Egg cases. FIGS. 3, 4. Egg case enclosed by soil particles; FIG. 5 Egg case disturbed to show the arrangement of eggs; FIG. 9. Individual eggs. FIGS. 6-8, 10-11. nymphs; FIG. 8. second stage nymph; FIG. 11. third stage nymph; FIG. 10 fourth stage nymph; FIG. 6. fifth stage nymph; FIG. 7. sixth stage nymph. FIGS. 12, 13 Adult grasshoppers. FIG. 14. Clearly shows that the female hopper can extend its abdomen to six inches soil level for oviposition as indicated by a scale.



S. VENUGOPAL

1. Shows the abdomen of female hopper trailing below the soil surface. 2. Retraction of abdomen after the process of egg laying. 3. Just hatched nymph. 4. First stage nymph. 5. Terminal abdominal portion of male grasshopper. 6. Terminal abdominal portion of female grasshopper. dv: dorsal valve of the ovipositor. vv: ventral valve of the ovipositor. c: cerei. sgp: sub-genital plate.

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Physiology of Disease in Plants *

BY

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(Received for Publication, 1958)

I feel it is my duty, at the very outset, to thank this University for having done me the great honour of asking me to deliver this series of lectures under the Dr. Todla Ekambaram Endowment. I was one of the last links in the chain of students admitted into the Botany Honours Course at the Presidency College, Madras by the late Professor Ekambaram and I am, therefore, particularly happy that I have been given the opportunity to deliver these lectures. The subject I have chosen is one in which I have been keenly interested for some years, namely, Physiology of Disease in Plants.

There is no doubt that there is today a great and urgent need to intensify both teaching and research in the functional mechanisms of living, healthy plants; there is also an equally urgent need to devote attention to problems relating to derangement in functional mechanisms such as are met with in a diseased plant. This subject is a most fascinating one and has received considerable attention from many workers abroad. In our own country, a great deal of fundamental work on the subject has been carried out in this University under the distinguished leadership of my teacher, Professor Sadasivan.

The problem of disease in plants is one of cause and effect. Causes there are many. Lack of nutrition is a common cause. This results in what are called "deficiency" diseases. The "grey speck" disease of oats which is due to manganese deficiency and the zinc deficiency of citrus are well-known examples. These are remedied by supplying the plants with the deficient element(s)

* Dr. Todla Ekambaram Endowment Lectures, 1957-58, delivered at the University of Madras on 17th and 18th October, 1958.

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in suitable doses, for instance, by foliage sprays. Other causes may be of an infectious nature and hence transmissible. Infections by fungi, bacteria, actinomycetes and viruses come under this category. In the account I shall now give, I shall be dealing largely with infectious plant diseases, especially those caused by organisms such as fungi and bacteria.

Before we consider the physiology of disease in plants, it will be useful to first deal briefly with the external manifestations of such disease, for such manifestations depend on derangements in functional physiology. In many soft and dry rots of plants, such as those of potatoes and several vegetables, whether they are caused by fungi or bacteria, we have a breakdown of the middle lamellae of cell walls followed by what may be termed a necrosis. Necrosis denotes killing of tissue. In such dry rots and fruit rots, the fruits usually become mummified and shrunken. Wilting is another common symptom in several diseases and this is clearly related to the water relations of the plants. Whether this is due to derangement in water absorption or transpiration or both is more than I can say, but as we shall see later, each individual disease will have to be considered on its own merits and there is little scope for generalisation. Chlorosis is another frequent symptom and is no doubt linked up with derangement of the photosynthetic mechanism or even uptake of essential ions. Hypoplastic effects such as stunting are also frequent and denote a reduction in plant vigour. Sterility accompanies disease quite frequently and we can even think of repercussions on the vital hormonal mechanisms here. There are then diseases which lead to the formation of "galls" which are in effect products of hypertrophy and hyperplasia.

Physiology of Infection

Whatever may be the external symptom picture, all infectious disease results from effective infection. We shall, therefore, first deal with the physiology of infection.

What is infection? Infection may be defined as the conveyance of a disease germ from outside into a host (Gäumann, 1950). In most diseases due to fungi, spore germination precedes infection. Indeed, it is a prerequisite for infection. There are many factors which govern spore germination and therefore govern infection also. Suitable moisture, pH, temperature, oxygen supply and the presence of nutrilites are some of the factors. A

detailed discussion of the physiology of spore germination is given by Gottlieb (1950).

Spores of some fungi, e.g., *Sclerotinia fructicola, Plasmopara viticola, Cylindrocladium scoparium,* and teliospores of *Gymnosporangium juniperi-virginianae* germinate only in contact with liquid water; spores of some other fungi germinate on dry surfaces in an atmosphere of high relative humidity, e.g., *Venturia inaequalis, Ustilago nuda.* A third group of fungi, largely represented by the powdery mildews, have spores which are able to germinate at extremely low relative humidity. It is obvious that the mechanism for germination under these very dry conditions is quite different from that of other spores which require liquid water and high humidity for germination. The "apparent osmotic pressure" of the cell sap of the conidia, according to Brodie (1945) is about 63 atm. for *Erysiphe polygoni* and 68 atm. for *E. graminis hordei.* Lilly and Barnett (1951) suggest that these high osmotic pressures may be an aid in absorbing moisture from a relatively dry atmosphere. According to Yarwood (1957) the explanation is probably the high water content of the conidia and their extremely efficient system of water conservation. Surface dry conidia of *Erysiphe polygoni* contain about 70% water, whereas other representative wind-borne fungal spores contain only about 10% (Yarwood, 1950).

Acidity is not usually a limiting factor for spore germination under natural conditions and fungal spores, in general, germinate over a wide range of pH. However, depending on the other conditions, pH may be a modifying factor in spore germination.

Temperature is one of the most important external factors governing germination. It may affect the percentage germination, the time required for germination and sometimes even the mode of germination. The effect of temperature on mode of germination is seen in the case of *Phytophthora infestans* in which below 20°C. the sporangia germinate more frequently by the formation of zoospores, whereas above 20° production of a germ tube is more frequent. It is not clear why different fungal spores should have different minimum, optimum and maximum temperatures for germination, but it is likely that favourable temperatures may stimulate certain enzymatic activities essential for germination.

Germinating spores respire vigorously and therefore an adequate oxygen supply is a prerequisite for germination. As with

temperature, supply of oxygen may even influence mode of germination. For example, sporangia of *Phytophthora infestans, P. colocasiae, P. palmivora* and *P. parasitica* germinate by producing zoospores in the absence of oxygen; germination by germ tubes was possible only when oxygen supply was present. On the contrary, oxygen appears to be essential for zoospore formation by sporangia of *Albugo candida, Plasmopara viticola* and *Sclerospora graminicola* (Uppal, 1926).

The spores of some fungi, e.g., *Uromyces caryophyllinus,* germinate best in pure water but those of *Aspergillus niger, Penicillium glaucum,* etc., need additional nutrients. The spores of *Coprinus* spp. are inhibited in the presence of sucrose, although 100% germination of spores of *C. micaceous* is seen in bean decoction (see Duggar, 1901). In the case of *Glomerella cingulata,* the spores require C, Mg, N and P for proper germination, but sulphur is not essential (Lin, 1945). Ryan's (1948) observation that the amino acids leucine, lysine, and proline favoured spore germination in mutants of *Neurospora* deficient for these amino acids shows that spore germination in some vitamin-deficient fungi may be stimulated by addition of the particular vitamins. Indeed, spores of *Rhizopus suinus* require meso-inositol for spore germination (Gäumann, 1950). The spores of some fungi such as *Botrytis cinerea* (Brown, 1922) and *Colletotrichum lindemuthianum* (Leach, 1923) germinate better in contact with plant tissue than in plain water. In these cases it has been suggested that certain substances diffuse out of the host plant into the infection drop containing the spores and stimulate germination and even infection.

Apart from physical conditions which favour germination of spores of plant pathogens, there is a good deal of evidence in favour of accepting "biological stimulation of germination" as a very common phenomenon. This is best illustrated with examples of some soil-borne pathogens. It has been shown, for instance, that germination of spores of *Urocystis tritici* is stimulated by roots of nonsusceptible plants such as pea, bean and rye and also traces of benzaldehyde, salicylaldehyde, acetone and butyric acid (Noble, 1924). Similar stimulation of resting spore germination of *Plasmodiophora brassicae,* the club root organism, is brought about by several crucifers and non-cruciferous species resistant to club root (Macfarlane, 1952). In contrast to the examples just cited, we may perhaps also distinguish what may be termed "biological

depression or retardation" of germination and growth. Varieties of flax resistant to Fusariose wilt are known to excrete hydrocyanic acid through their root systems and this is known to depress the growth of the pathogen (Timonin, 1941). More recently, Buxton (1957a, b) has shown that root exudates of pea varieties susceptible to Fusariose wilt stimulate spore germination of the pathogen, but those of resistant varieties inhibit this.

We shall now consider the mode of entry of the pathogen and how entry into the host is effected.

In many cases, direct penetration is common, e.g., *Colletotrichum lindemuthianum, Fusarium niveum, Diplocarpon rosae, D. earliana, Taphrina deformans, Pseudopeziza ribis, Ustilago zeae, Venturia inaequalis, Erysiphe polygoni, Sclerotinia sclerotiorum, Corticium rolfsii, Bremia lactucae* (conidia), *Maravalia hyalospora, Cronartium ribicola* (sporidia) and *Sclerospora graminicola.* In certain others, stomatal penetration is the mode, e.g., *Mycosphaerella fragariae, Plasmopara viticola, Cladosporium fulvum, Maravalia hyalospora* and *Bremia lactucae* (zoospores). Whilst direct penetration or stomatal penetration is obligatory for many fungi, for a few it is not obligatory, e.g., *Maravalia hyalospora.* In still others, entry may be effected by different avenues, e.g., *Bacterium campestre* through hydathodes, *Glomerella cingulata* through leaf scars, *Phytophthora infestans* through lesions of powdery scab, *Agrobacterium tumefaciens* through insect wounds, *Corynebacterium tritici* through nematode injury and *Erwinia amylovora* through stigmas, anthers and stomata. The conidia of *Nectria galligena* which causes cankers in trees are sucked into the tracheids exposed by leaf fall. In the case of the smuts, *Tilletia tritici* and *Ustilago violacea,* seedling infection is usual. An account of the modes of infection by soil-borne pathogens is given by Sadasivan & Subramanian (1959).

In the case of fungal infections, e.g., infection of leaves of *Vicia faba* by *Botrytis cinerea,* stimulation of the pathogen by exosmosed substances has been postulated. It has been shown that in distilled water with a conductivity of 0·8 units, the germ tubes of the fungus spores grow very slowly, but an enhanced growth rate of the germ tubes is seen when there is a rise in conductivity of the drop which is due to an increase in the materials dissolved in it. The electrical conductivity is taken as an

index of the salt content of the water drop which is modified by the exosmosed substances of the host.

Kusano (1936) made a study of the parasitism of *Olpidium* on *Vicia* and indicated that positive chemotaxis of the swarm cells to potassium is the first step in the invasion of the host and that chemotactic and chemotropic responses of the parasite are of primary importance in parasitism. That "exosmosis" of substances on leaves occurs is now admitted. The work of Arens (1934) is of particular interest here. He showed that distilled water (pH 5·6) sprayed on leaves became alkaline in a few hours, considerably more so in bright light than on shaded plants. Lower alkalinity of the water on shaded plants was attributed to their greater respiration, the carbonic acid formed partially neutralizing the exosmosed alkaline salts. The alkalinity of the water on leaf surfaces is caused by exosmosis of potassium, calcium, magnesium and phosphorus as well as organic substances. Of these, potassium, in particular, is excreted in large quantities. Experiments with several species showed that the mineral content of the leaves could be reduced in twenty-four hours by 50% through exosmosis. It has further been shown recently that exosmosed substances on paddy leaves include glycine, alanine, methionine, tryptophane, lysine and succinic acid (Subba-Rao & Suryanarayanan, 1957). Kovacs & Szeoke (1956) discussed the inhibitory/stimulatory influence of cuticular excretions (by exosmosis) of wheat, red clover, poplar and several other plants on the germination of spores of *Botrytis cinerea, Puccinia triticina* and other fungi. Hafiz (1952) recorded secretion of malic acid by gram (*Cicer arietinum*) leaves and the greater amounts of malic acid secreted by the plants resistant to *Mycosphaerella* blight was thought by him to inhibit spore germination. The work of Topps & Wain (1957) clearly brought out the fungistatic properties of exudates from leaves of ash, beech, chestnut, elder, *Robinia pseudacacia*, hazel, etc., in retarding growth of germ tubes of *Botrytis cinerea*. Arens' (1934) observations and those of the others cited above have been strengthened by the work of Long, Sweet & Tukey (1956) who have shown clearly using radioisotopes that organic and inorganic nutrients are lost from plant foliage by leaching. Loss occurs throughout the growing season, increasing greatly just before maturity and death of the foliage. The rate of loss is governed by factors such as light, nutrient level, maturation of leaves, etc. Large quantities of reducing substance(s) and ninhydrin-positive

materials (polypeptides?), as well as potassium, calcium, sodium and phosphorus have been identified in the leachates.

The question may be raised: is there a stimulus to penetration and, if there is, what is it? Stakman & Harrar (1957) have discussed this question lucidly. Miyoshi (1895) did pioneering work in this field and, working with different types of membranes, fungal spores and chemical solutions, concluded that the stimulus to penetration was positive chemotropism. Good support to Miyoshi's views came from Massee. Clarke & Fulton did similar work but indicated that (1) the effect of water was as pronounced as that of presumably attractive chemicals; (2) chemotropic responses of fungi did not discriminate between those that were nutritive and those that were deleterious such as copper sulphate; and (3) germ tubes often showed negative chemotropism, i.e., the tendency to grow away from their own metabolic products. Much evidence against a generalized positive chemotropism theory came from the critical work of Brown (1936, 1948). Brown's work with *Botrytis cinerea* showed that the fungus penetrated strips of onion epidermis which had been washed several times and hence free of diffusible substances. The hyphae could grow from the inner to the outer surface and *vice versa* with equal facility; further, regardless of original distribution of nutrient materials, membranes of formalized gelatin were penetrated. Dickinson (1949) made a study of eleven species of rusts and six species of powdery mildews with special reference to behaviour of germ tubes. He concluded that the stimuli affecting germ tubes in his experiments were positive hydrotropism (growth toward moisture or water), negative thigmotropism (growth away from a solid surface) and dia-thigmotropism (growth along a surface) and that the nature of the host surface would affect the thigmotropic response very considerably. The production of appressoria by many fungi, e.g., *Colletotrichum phomoides* (Van Burgh, 1950), *C. linicolum* (Schwinghamer, 1954) and by *Helminthosporium victoriae* (Paddock, 1953) is a case of thigmotropic response and such fungi tend to produce appressoria on many types of hard surfaces, e.g., glass, mica, etc., which they cannot penetrate. As Stakman & Harrar (1957) have stated, the stimuli to penetration are varied and complex, and it is not possible yet to establish any basic principles.

Another interesting observation worth mentioning here is the fact that in any given fungus the mode of penetration would

depend on the nature of the fungus itself. For instance, infection by sporidia of *Puccinia graminis* takes place by direct penetration but that by aeciospores and uredospores takes place through stomata. It is not clear if the mode of penetration in this case is dependent also on the nature of the host. However, in the case of *Bremia lactucae,* which causes the downy mildew of lettuce, conidial germ tubes penetrate directly and in the few cases when the sporangia germinate producing zoospores, penetration is through stomata. Thus, infection of the same host by the same pathogen is achieved differently by the differing behaviour of the asexual stage. Direct penetration is obligatory for the sporidia of rusts, stomatal penetration for aeciospores and uredospores; but it is variable for pathogens such as *Bremia lactucae,* although the variability is in response to the differing behaviour of the asexual stage.

Yet another aspect which deserves special mention in any discussion of infection phenomena is the osmotic pressures of host and parasite. Thatcher (1942) made a critical investigation of osmotic and permeability relations in parasitism. His results clearly indicate that an osmotic pressure higher in the fungus cells than in the surrounding host cells is characteristic of the host-parasite relationship. This is logical and essential before the pathogen can absorb water from the host cells. Indeed, osmotic pressure of the substrate is always a limiting factor for growth of pathogens even in pure culture. A further fact which emerges from Thatcher's work is that there are no significant differences between the osmotic pressures of cells of obligate and facultative parasites, relative to the susceptible host cell.

It is noteworthy that even in the case of infections by phanerogamic parasites, a similar situation is met with. For example, in experiments in plasmolysis, Lilienstern (1932) observed that haustoria of *Cuscuta monogyna* had a slightly lower osmotic pressure than the cortex of *Melilotus albus,* a resistant host, and a significantly higher osmotic pressure than the cortex of *Cicer* sp., a susceptible host.

The nuclear status of the spore type which infects the plant is especially important in the case of heteroecious rusts in which, for example, the haplophase is usually found infecting dicot hosts, whereas the dikaryophase infects and perpetuates itself on various grasses.

Symptom pictures in relation to host physiology

I shall now attempt to discuss briefly the symptom pictures of plant disease in relation to their derangement in host physiology. As already mentioned, every manifestation of disease should have its physiological basis, for form and function go hand in hand. In other words, we shall go a little deeper into the question of cause and effect in infectious plant disease.

NECROSIS

Mention has been made of killing of tissues or necrosis in several soft rots and dry rots of fruits, vegetables and tubers and we may include under this category leaf spots and blights as well. Damping-off of seedlings and various soft rots are associated with breakdown of the cell walls and a maceration effect resulting from separation of the individual cells from each other. Maceration usually leads on to death of the protoplasts and the macerating effect is usually seen to take place in advance of the pathogen. One of the earliest investigations on the pathology of soft rots was that of de Bary (1886) on *Sclerotinia libertiana*. de Bary showed that an enzyme secreted by the fungus caused dissolution of the middle lamella of cells near the parasite. He attributed cell-killing to oxalic acid which was shown to be produced by the fungus *in vitro*. Much work has been carried out by Brown (1934, 1936) and his school on this problem using *Botrytis cinerea* and it has been shown that this fungus macerates plant tissue by secreting pectic enzymes and that inorganic substances play no part in this process. The death of the protoplast is either the direct result of enzyme action or is caused by a toxin which functions only after the tissue has been macerated. Indeed, pectic enzymes are now considered to play a significant role in plant infections by facultative pathogens since pectin itself is a major constituent of plant cell walls. The enzymes which help in this are of the pectolytic type, viz., pectin methyl esterase (PME), polygalacturonase (PG) and depolymerase (DP). Pectin-esterase is commonly produced by many micro-organisms and also occurs in higher plants; it catalyses the hydrolysis of the methyl-ester groups in pectinic acids and pectin to give methyl alcohol and pectic acids or pectinic acids of lower methoxyl content. PG hydrolyses the 1, 4-glycosidic linkages between adjacent anhydrogalacturonic acid residues with profound effects on the properties of the chains.

It is produced by many microorganisms but has been reported from higher plants very rarely. Essentially, the substrate of this enzyme is pectic acid; it is more active on pectinic acids of low methoxyl content. On the other hand, DP attacks highly esterified pectinic acids, in some cases more readily than low methoxyl pectins. The pectic enzyme which is of great interest is protopectinase whose substrate is the ill-defined protopectin and which is usually distinguished by its action on living tissue. The protopectinase of a number of facultative pathogens has been investigated by several workers. Some pathogens (e.g., *Bacterium aroideae, Pythium debaryanum*) form active solutions in the absence of pectic substrates; *Botrytis cinerea* and *Verticillium dahliae* produce active preparations without pectic substrates, but when these are present the activity of the preparations is definitely greater. Still others, e.g., *Sclerotium rolfsii* and *Fusarium moniliforme* produce protopectinase only when pectic substrates are present. The enzymes from different pathogens differ both in regard to their pH optima as well as their response to heat treatment. A careful consideration leads one to conclude that there is no uniformity in the end-products of pectic substances degraded by various organisms.

In regard to the action of pectic enzymes on living cells, Brown (1915) stated that there were two possibilities: (i) the death of the cells after maceration was brought about by one or more substances responsible for cell separation; (ii) that the killing substance may be quite distinct, but was unable to act upon the protoplast until this had been modified in some way by the action of the macerating enzymes. In the case of *Botrytis* at least, it has not been possible so far to separate a cell-killing from a cell wall dissolving substance and it may be concluded provisionally that wall dissolution and lysis are but stages of a progressive sequence of events brought about by enzymatic activity (Brown, 1948). In a discussion of pectic enzymes and plant disease, Wood, (1955, p. 283-84) states: "results have shown that a considerable number of treatments affecting the rate of maceration have a similar effect on the rate at which cells are killed; this might be taken as circumstantial evidence that it is the action of the protopectinase itself which kills the cell. There is some evidence that maceration increases the susceptibility of cells to simple toxins; maceration may remove the pectic substances between the cellulose microfibrils and therefore aid the movement of the toxins through the cell

wall. But even in the absence of maceration mercuric chloride kills cells quite quickly, whereas the postulated toxin in the protopectinase preparations does not. The difficulty of attributing a toxic action to a protopectinase or any other pectic enzyme is that these enzymes, by their very nature, must be considered specific in their action, and at present their substrates are considered to be restricted to the cell wall and the cell vacuole. If protopectinase acts only on inert wall material, it is difficult to see why cells should be so rapidly killed, for if the tissue under test is allowed to macerate undisturbed there seems no reason why the plasmodesmata should not remain intact, the cytoplasm merely being now limited by a more permeable cell wall. On the other hand, if pectic compounds play some part in the relation between the innermost layer of the cell wall and the outermost layer of the cytoplasm, degradation of this layer might well affect the vital properties of the protoplast. As far as the writer is aware, no one has ascribed a special role to pectic materials in the structure of the outer protoplasmic layer which is generally considered to be a lipoid-protein complex. The assumption of a boundary layer containing pectic substances does little to explain why plasmolysis increases the resistance of cells to killing. This remains one of the most puzzling features of the work described. Even if protopectinase acts upon a special pectic layer at the protoplast surface, it is difficult to imagine why the retraction of the cytoplasm from the cell wall should make this layer immune to attack. If toxicity is caused by a specific toxin, it is possible that the change from the unplasmolysed to the plasmolysed state may greatly affect the rate at which toxins pass into the cytoplasm.

"At present, therefore, there can be no complete understanding of the way in which protopectinase acts upon living cells, and it is obvious that the above work needs to be repeated with much purer enzyme preparations to reduce the possibility of misinterpretation, which is always present when crude preparations are used. It is apparent, however, that the results so far obtained indicate that the relation between the outer layers of the cytoplasm and the innermost layer of the cell wall may be quite complex and that the study of it may be important for a fuller understanding of the way in which toxins enter and kill cells" (Wood, 1955, p. 283-284).

Although the secretion of pectic enzymes appears to be an important factor in plant disease, the ability of an organism to secrete

these enzymes in pure culture may not be related to its pathogenicity.

It would appear that one of the main factors influencing pathogenicity of these organisms is the rate at which they grow and secrete protopectinase relative to the rate at which the host tissue reacts to form surface layers resistant or immune to attack. Another factor affecting pathogenicity is the presence of factors inhibiting pectolytic activity in the host plant sap. Brown (1915) showed that slices of potato tuber soaked overnight in an extract of leaves of broad bean (*Vicia faba*) became resistant to the action of protopectinase from *Botrytis cinerea*. Similarly, Cole (1953) reported that oxidized extracts from several plant tissues markedly inhibited activity of protopectinase and depolymerase from *B. cinerea,* but this effect was not so well seen in the case of enzymes of *Monilia fructigena*. A thermolabile inhibitor of pectinase has also been shown to be present in pear fruit (Weurmann, 1953). Inhibition of pectic enzymes may well be the basis for disease resistance in such cases.

Another point of interest here is the effect of carbon/nitrogen ratio of the substrate on enzyme activity. In general, high ratios tend to increase mycelial growth, whereas lower ratios give poorer growth and lead to a great increase in enzyme secretion. The significance of this is obvious from the work of Vasudeva (1930) who found that *Botrytis allii* is normally not parasitic on apple fruit, but invaded this tissue when a source of nitrogen was added to the inoculum.

We may consider here also *in vivo* enzymatic changes leading to symptoms characteristic of brown rots such as are seen in apples infected by *Sclerotinia fructigena*. Active PG and PME are produced *in vitro* by the pathogen. Cole's (1958) recent investigation into the reasons for the presence of a firm brown rot and the absence of PG activity in tissues of seedling apples attacked by the fungus suggested that the fungus does secrete the enzyme, which attacks the pectic materials of the host tissue; but the fungus also produces polyphenoloxidase which, together with the same enzyme from the host, oxidises the leuco-anthocyanins to a polymer which inhibits the PG activity possibly by a tannin reaction, before it has time to macerate the tissue and produce a soft rot. The oxidation of the leuco-anthocyanins, catechins, and other compounds causes the brown discolouration.

LEAF SPOTS

In the case of certain leaf spot diseases with characteristic localised necrotic lesions or extensive spots resulting from coalescence of such lesions, the disease syndrome appears to be brought about by specific toxic substances secreted by pathogens *in vivo*. The leaf blight of oats caused by *Helminthosporium victoriae* is a good example. This fungus causes a local necrosis on roots but the diseased plants show a conspicuous red striping of the leaves. No fungus could be isolated from spots which is suggestive of the fact that the stripes are toxigenic in origin. A toxin (polypeptide) isolated from *in vitro* cultures of the fungus reproduces on healthy plants the leaf striping typical of the disease. It has not been shown to be produced *in vivo* but extracts from diseased leaves are phytotoxic but not those from healthy plants (Brian, 1958). Pringle & Braun (1958) have reported that the toxin from *H. victoriae* is very unstable. Two materials which reacted with ninhydrin as the preparations lost their activity were separated, one an apparently undescribed compound (victoxinine) with the empirical formula $C_{17}H_{29}NO$, and the other probably a peptide. Victoxinine was shown to inhibit root growth at concentrations of $2{\cdot}5 \times 10^{-4}$M and it was suggested that specificity may be a function of the peptide part.

In early blight of potatoes and tomatoes due to *Alternaria solani* lesions are produced on leaves, stems and fruits and the fungus can usually be isolated from such lesions. Occasionally, however, when the primary infection is at the base of the young plant, stem and leaf lesions appear to be sterile; similarly, when primary infections are on petioles, sterile lesions may be seen on leaves. These findings are suggestive of toxin production at the focus of infection and its movement from there to different parts of the plant in advance of the actual spread of the pathogen (Brian, 1958). There is good reason to believe that alternaric acid which is a metabolic product of *Alternaria solani in vitro* and is an optically inactive unsaturated dibasic acid (mol. formula $C_{21}H_{30}O_8$) may be implicated in the etiology of the disease. Definite proof that alternaric acid is produced *in vivo,* however, is not available and the fact that virulent strains of the pathogen do not yield the metabolite *in vitro* suggests that other toxins may be involved (Brian, 1958).

In the case of tobacco wildfire caused by *Pseudomonas tabaci,* small angular necrotic spots are produced on leaves and each spot

is surrounded by a distinct chlorotic halo. The pathogen can be isolated from the necrotic spots, but not from the surrounding chlorotic haloes. If cell-free culture filtrates are injected into leaves, they cause chlorotic areas. This is suggestive of the fact that the chlorotic areas are toxigenic in origin and this suggestion is, indeed, strengthened by the fact that attenuated strains of the pathogen which have lost their ability to produce the toxin *in vitro,* were not able to cause chlorotic haloes but were able to cause necrotic spots thereon. The toxin produced *in vitro* has been isolated and shown to be α-ε-diamino-β-hydroxypimelic acid and is now considered to interfere with methionine metabolism of the leaf. In this case also, however, its production *in vivo* has not been definitely proved (Brian, 1958).

The disease syndrome in the case of die-back and shot hole of stone fruits due to *Pseudomonas mors-prunorum* again is considered to be induced by a toxin produced *in vivo* and tissues frequently die well in advance of actual bacterial invasion. The toxin is probably a protein endotoxin, liberated into the medium on autolysis of the bacterial cells; its production *in vivo* has not been definitely demonstrated (Erikson & Montgomery, 1945).

VASCULAR WILT DISEASES

I shall now pass on to a consideration of 'wilting' which is a symptom characteristic of many diseases; but I shall deal here mainly with the so-called 'vascular wilts', problems relating to which have received much attention in recent years. Vascular wilts may be caused by bacteria or by species of fungi, especially those of the genera, *Fusarium* and *Verticillium.* The disease syndrome in vascular wilts includes epinasty and root development in the formative period and leaf wilting and vascular discolouration during the later stages.

The cause of epinasty in tomato wilt (caused by *Fusarium oxysporum* f. *lycopersici*) is presumably ethylene which is produced in sufficient amounts both *in vitro* and *in vivo* by the fungus. It has been pointed out that ethylene may also stimulate adventitious root formation (Dimond & Waggoner, 1953b).

There is no doubt that disturbances to the water economy of the plant follow infection by wilt-pathogens. Injury to root systems soon after infection is infrequent in most vascular wilts and death of root systems does not take place until after the plant wilts and

collapses; impairment of water-solute absorption by infected root systems during early phases of the disease is therefore a remote possibility (Dimond, 1955; Ludwig, 1952). According to Gäumann (1951) wilting of tomato plants infected by *Fusarium* is attributable to lycomarasmin, a "wilting toxin" produced by the pathogen; lycomarasmin would have a coagulating effect on the plasma as a result of which the semi-permeability of the plasma membrane is damaged and the osmotic prerequisites for turgor are destroyed. A part of the plasmatically bound water is also released, leading to an excess in the transpiration rate. Wilting caused by lycomarasmin, by its very nature, is irreversible, whereas drought-induced wilting is reversible. On the other hand, several workers have suggested impaired water transport through the xylem as an explanation for wilting (Ludwig, 1952; Scheffer & Walker, 1953). Dimond & Waggoner (1953a) measured the velocity of the transpiration stream in diseased and healthy cuttings by means of radiophosphate movement. The rate of movement of radiophosphate was 0·0045 cm./sec. where disease was severe and 0·0091 cm./sec. when the stem was moderately diseased. In healthy stems, under comparable conditions, the rate of movement of solutes was 0·25 cm./sec. Thus the rate of movement in diseased plants is only 2-4% of that in healthy plants. A wilting leaf from an infected plant regained its turgor when discoloured vascular tissue in the petiole was first removed and the basal end of the remaining petiole tissue was then placed in water; it is only necessary to remove the dysfunctional tissue in order to observe the unimpaired osmotic properties of cells in wilted leaves from diseased plants — an observation which lends little support to Gäumann's hypothesis of irreversible destruction of the osmotic prerequisites for turgor.

The basic difference between Gäumann's hypothesis and that of the other workers lies in the fact that the former signifies impairment of water uptake and transpiration due to the action of lycomarasmin on host cells, whereas the latter suggests impairment of transpiration alone which is the result of impaired water flow. Decreased transpiration rates which have been demonstrated for tomato wilt by Dimond & Waggoner (1953a) have been recorded for Fusariose wilt of cotton also (Subba-Rao, 1957a). A critical evaluation of the results obtained by various workers leads one to conclude that in wilt diseases there is a definite impediment to water conduction through the xylem. This is claimed to be brought about either by some homogeneous hyaline material in

the vessels of the infected plants (Ludwig, 1952), or polysaccharides produced by the pathogen (Hodgson, Peterson & Riker, 1949), or conjugated phenols arising from hydrolysis of β-glucosidases in the host (Davis, Waggoner & Dimond, 1953), or again products resulting from the maceration of cells in the xylem due to the action of exo-cellular pectic enzymes like PME (Winstead & Walker, 1954). As early as 1926, Tochinai suggested that production of carbon dioxide by the pathogen in tracheae caused a gas lock in the vessels, but there is little evidence to support this gas embolism theory. Dimond & Waggoner (1953a) could not observe any difference in the viscosity of tracheal fluids of *Fusarium* infected tomato plants, so that it is clear that impediment to water flow may not be the consequence of increased viscosity. All the available data on the extent of occlusion of vessels by fungus mycelium and rate of flow indicates that the reduction in flow due to mycelial plugging is probably less than 5% (see Dimond, 1955).

It will be pertinent to consider here the comparative merits of the two schools of thought which hold divergent views on the mechanism of plant wilts. The American workers led by Walker at Wisconsin and by Dimond & Waggoner at Connecticut have presented coherent evidence pointing to "vessel plugging" as the most plausible explanation. In a general way, this point of view has been summed up by Dimond (1955, p. 346-47) as follows: "vascular discoloration appears to result from melanin production in living cells. Melanin is produced through the oxidation of polyphenol by terminal oxidases. The polyphenols appear to arise from hydrolysis of glucosides by β-glucosidase. β-glucosidase in the host cell or that produced by the pathogen may be involved. In either case, the reaction may be initiated through disorganization of cells, brought about by the action of fungal pectic enzymes. In the case of hosts which lack such oxidases in xylem parenchyma or pathogens which do not form a β-glucosidase, no discoloration develops, e.g., watermelon wilt.

"The wilt producing pathogens produce pectic enzymes which partially or completely hydrolyze pectins exposed at pits in vessels. The hydrolytic products can form calcium gels if partially hydrolyzed or gums if acted upon in turn by other enzymes. In either case, plugging of vessels and water shortage in leaves develops and becomes more acute with time. As melanoid pigments are released into the vascular elements, the pigment may be trapped by the gels, contributing to vascular discoloration. Alternatively, the

gums may polymerize as they age and become darker in color. Vascular discoloration may thus result from more than one process".

The toxin theory of plant wilts has received support largely from the Swiss school of workers led by Gäumann. A major part of the Swiss work is also on tomato wilt and, as already mentioned, wilting is attributed to the action of lycomarasmin. Lycomarasmin is produced by *Fusarium oxysporum* f. *lycopersici in vitro*: it is a simple dipeptide formed from asparagine, glycine and α-hydroxyalanine, has a molecular weight of 277 and an empirical formula of $C_9H_{15}O_7N_3$. On tomato cuttings, lycomarasmin causes the margins of leaflets to roll upwards and become brittle and withered; the cuttings develop symptoms of transpirational imbalance leading to wilt, although this is not confirmed by certain workers. The toxicity of lycomarasmin is increased in the presence of iron salts with which it is considered to form a chelate complex. A serious objection to assigning any role to lycomarasmin in tomato wilt is the fact that its production or presence *in vivo* in infected plants has not been proved. Unfortunately, the substance of Gäumann's hypothesis has been built up on results obtained from work on *in vitro* culture filtrates of the pathogen and their effects on cut shoots. The difficulties in accepting lycomarasmin complicity in tomato wilt are: (i) differences in intensity of disease between normal and Fe-deficient plants, such as would be expected if the Fe-lycomarasmin chelate were important, could not be demonstrated (Scheffer & Walker, 1953); (ii) preferential chelation of cupric ions with lycomarasmin renders the lycomarasmin-Fe complex unstable and the Cu-lycomarasmin chelate is much less phytotoxic; Fe-lycomarasmin is also unstable in the presence of 8-quinolinol, which produces a more stable iron complex than Fe-lycomarasmin. However, no protection to artificially infected plants is seen when they are treated with copper salts or 8-quinolinol (Waggoner & Dimond, 1953); (iii) in culture filtrates, lycomarasmin appears in quantity only after autolysis has set in and therefore its production *in vivo* in sufficient quantity in the period (about 15 days) between inoculation of a plant and wilt development may be unlikely. The results of work on lycomarasmin and its phytotoxic effects do not, therefore, lend support to the toxin theory.

However, the work done at Madras under the leadership of Sadasivan (see Sadasivan, 1958), especially on cotton wilt, appears

to bring in evidence in favour of Gäumann's hypothesis, viewed in juxtaposition with work done elsewhere on fusaric acid which is one of the metabolic products of *Fusarium vasinfectum* which causes the vascular wilt of cotton in India. Early work on this problem was of the conventional type and toxicity of dialysed culture filtrates of *Fusarium vasinfectum* to cut shoots of cotton was demonstrated (Kalyanasundaram & Lakshminarayanan, 1953) and it was also shown that the dialysed culture filtrates contained also growth factors stimulating rooting of cut shoots (see Subramanian, 1955). Subsequently, presence of fusaric acid *in vivo* in cotton plants infected by the pathogen was demonstrated using a chromatographic method wherein the metabolite was identified as its Cu-chelate (Lakshminarayan & Subramanian, 1955). This has since been confirmed by the Swiss workers (Gäumann, 1957). Fusaric acid was first isolated by Yabuta, Kambe & Hayashi (1934) as a metabolic product of *Fusarium heterosporum,* but since then it has been shown to be produced *in vitro* by *F. oxysporum* f. *lycopersici, F. vasinfectum, F. orthoceras* and *Gibberella fujikuroi* (Gäumann, 1957; Venkata Ram, 1957; Sadasivan, 1958). Chemically, it is a pyridine-carboxylic acid (5-butylpicolinic acid) with a molecular weight of 179, an empirical formula $C_{10}H_{13}O_2N$ and a melting point of 98-100°C. In culture filtrates of *Fusarium oxysporum* f. *lycopersici* and *Gibberella fujikuroi,* following the appearance of fusaric acid, dehydrofusaric acid is produced. Dehydrofusaric acid (5-butylenicpicolinic acid) has the empirical formula $C_{10}H_{11}O_2N$, a molecular weight of 177 and a melting point of 118-120°C. Like lycomarasmin, fusaric acid forms chelate-complexes with heavy metals such as iron and also causes wilting and leaf necrosis. Fusaric acid is known to be potentiated *in vitro* in bioassay in the presence of free $F^{\prime\prime\prime}$ (see Sadasivan, 1957). In a diseased plant fusaric acid chelation with iron may lead to iron deficiency and it is attractive to postulate the vein-clearing symptoms being linked up with deficiency of this important heavy metal, iron. This postulate derives some support from the observations of Varadarajan (1953) who found that addition of iron to the infested soil, alone or in combination with manganese, gave protection to cotton plants from wilt. Another observation which suggests a key role for fusaric acid in plant wilts is the fact that preferential chelation is possible here too and the theoretical expectation of the benefit of such chelation in giving plants protection from wilt is realised in controlled experiments. For instance, pre-treatment of cut shoots of cotton to 5-10

p.p.m. of 8-quinolinol gave effective protection from toxaemia due to dialysed culture filtrate of *Fusarium vasinfectum*. The chemotherapeutic action of 8-quinolinol is possibly through the removal of heavy metals *in vivo*, rendering them unavailable for chelation with the toxin (Subramanian, 1956). Since the chemotherapeutant was active only when the plants were pre-treated, it was suggested that the toxic metabolites have relatively higher chelating capacity than 8-quinolinol, while 8-quinolinol-heavy metal complexes are more stable *in vivo*. Lakshminarayanan (1955) detected high quantities of cystine in resistant cotton plants and none in susceptible ones; the mechanism of wilt resistance, according to him, is linked up in this case with iron chelation with cystine in the resistant plants. That this is likely is shown by the 'acquired' resistance of susceptible cotton plants to wilt when the soil is amended with zinc, for the zinc treatment led to greater amounts of cystine in susceptible plants, and preferential chelation of cystine with iron was considered to prevent fusaric acid-iron chelation. It is clear that heavy metals have a significant role in the physiology of plant wilts and the possibility of vivotoxins chelating with iron and thus robbing the plants of this essential ion should be examined. Ash analyses of diseased cotton plants showed, however, that there were greater amounts of iron in infected than in healthy plants (Sadasivan and Kalyanasundaram, 1956) and it would therefore be worthwhile ascertaining how much of this iron present in the host is mobile and available to the plant.

Spectrochemical analyses of cotton plants infected by *Fusarium vasinfectum*, using the standard Lundegårdh flame emission (spark-in-flame) method, showed an increased uptake of magnesium, calcium, iron and manganese with decreased accumulation of potassium over healthy plants (Sadasivan & Kalyanasundaram, 1956). Compared to the healthy plant, therefore, there is a definite derangement in the selective absorption of ions in the infected plant and such a derangement is more likely to have been caused by chemical agents (vivotoxins) than mere physical causes such as mechanical plugging of the conducting system. Further work (Sadasivan & Saraswathi-Devi, 1957) on this showed that, in comparison with healthy plants, there was an enormous rise in the amount of magnesium and manganese in the infected but apparently healthy plants. In the plants which were wilting also greater amounts of manganese were seen than in healthy plants.

On the other hand, apparently healthy infected plants had much less potassium, and wilting plants still less of the element, than healthy plants. In the resistant plants the ratios of the three elements potassium, calcium and magnesium remained unaltered, whereas in the susceptible variety these ratios decreased following infection. In view of the importance of these findings, the actual data obtained by Sadasivan & Saraswathi-Devi are reproduced in Tables I and II with the kind permission of the authors.

TABLE I*

Percentage loss or gain in elements due to infection in soil.

Element	Resistant	Susceptible	
		Apparently healthy	Wilting
Potassium	23·3 (—)	80·3 (—)	99·5 (—)
Calcium	24·5 (—)	29·1 (—)	57·0 (—)
Magnesium	23·7 (—)	95·0 (+)	38·1 (—)
Manganese	0	170·6 (+)	11·8 (+)

TABLE II*

Ratios between elements

Samples	Resistant		Susceptible		
	Healthy	Grown in inoculated soil	Healthy	Grown in inoculated soil, apparently healthy	Wilting
K : Ca	0·9	0·9	2·5	0·7	0·03
K : Mg	2·3	2·3	10·1	1·1	0·8
Ca : Mg	2·6	2·6	4·1	1·5	2·9
K : Mn	61·4	47·0	125·3	9·1	0·5
Ca : Mn	70·0	52·9	50·6	13·3	19·5
Mg : Mn	27·1	20·7	12·4	8·7	6·8

* after Sadasivan & Saraswathi-Devi (1957).

It is quite obvious from these results that even during early phases of disease of wilting cotton plants, there is a definite derangement in the uptake and accumulation of inorganic ions which is clearly the result of toxaemia. The results of conductivity studies of plant cells of healthy and diseased plants (Gnanam, 1956) also indicate increased accumulation of ions in the latter. It was shown by Subramanian (1956) that infected roots of cotton plants accumulated more iron than healthy ones, although compared to resistant ones the concentration of iron in susceptible roots was less. Although much more information seems necessary to collate these valuable data and obtain a coherent picture of the *in vivo* changes characteristic of vascular plant wilts, the results of ash analyses just referred to definitely show that mere mechanical impediment to water flow through the xylem cannot explain the sequence of changes within the host following infection by *Fusarium*.

It is noteworthy that there is a correlation between the period required for production of fusaric acid *in vitro* and the appearance of symptoms in infected plants—a correlation lacking in the case of lycomarasmin and tomato wilt (see p. 25). Fusaric acid appears in culture filtrates of *Fusarium vasinfectum* within four days of fungal growth and is produced in cotton plants soon after infection by the pathogen. Symptoms of fusaric acid toxicity can be seen in seedlings which are 13 days old; indeed, it is found *in vivo* in amounts up to 17 mg./kg. fresh weight (Kalyanasundaram & Venkata Ram, 1956; Gäumann, 1957).

Another line of evidence suggests that factors other than fusaric acid may have to be looked for to explain the disease syndrome in entirety. For instance, a study of tissue respiration of healthy and diseased cotton plants showed that infection is followed by increased respiration. Zinc amendment to soils brought about a decrease in respiration of infected susceptible plants; indeed, the respiratory rate of these plants was similar to that of healthy plants. However, the effect of fusaric acid on host tissues was quite different in that it inhibited tissue respiration (Lakshmanan, 1956). It is, therefore, clear that respiratory effects such as are seen in diseased plants cannot be due to direct fusaric acid toxicity.

Any substance or treatment which affects respiration might be expected to affect permeability since the structure of cytoplasm and cytoplasmic membranes is maintained by metabolic energy (Kramer, 1955). If this is accepted, then some of the changes

observed in ionic imbalance in diseased plants may be explainable as directly resulting from respiratory effects. Further, the evidence available on nonosmotic water uptake (see Kramer, 1955) indicates that by the expenditure of energy released in enhanced respiration, ions (and water) may accumulate in root cells nonosmotically. Indeed, Lundegårdh (1950) relates water uptake to respiration through ion uptake, the water either being carried along with the ions or moving along an osmotic gradient produced as a result of salt accumulation. Studies on the effects of respiratory inhibitors on healthy and infected plants may throw further light on this problem.

The lead the Madras school under Sadasivan has given in understanding the complex problem of fungal wilts is admirable and there is a great need to approach the problem of tomato wilt also from the same angle. The existing controversy regarding the mechanism of this wilt could perhaps be cleared up by looking for something other than lycomarasmin as the chief cause of the disease syndrome, until definite evidence for its presence *in vivo* is presented. Want of space prevents me from discussing the role of pectic enzymes in cotton wilt, for details of which the reader may refer to papers by Sadasivan (1957, 1958), Subramanian (1956) and Lakshminarayanan (1956, 1958a, b).

HYPERPLASTIC DISEASES

We shall now consider examples of plant diseases which lead to the formation of warts, galls, etc. Crown gall on various hosts caused by *Agrobacterium tumefaciens,* maize smut caused by *Ustilago maydis,* club root of crucifers caused by *Plasmodiophora brassicae,* potato wart caused by *Synchytrium endobioticum,* crown wart of alfalfa caused by *Physoderma alfalfae,* cedar-apple rust due to *Gymnosporangium juniperi-virginianae,* apple canker due to *Nectria galligena,* woody gall of acacia due to *Hapalophragmiopsis ponderosa,* etc., are well-known examples. Hypertrophy and hyperplasia which are characteristic of the disease syndrome in these cases are suggestive of increased auxin production and it will be interesting to consider the physiological effects of pathogenesis which lead to such tissue proliferation. The ability of various microorganisms to produce indole-acetic acid (IAA) *in vitro* has been established in a number of cases and the fact that it is a microbial metabolic product indicates for it a possible role in intiating cell division. As early as 1935 Thimann (1935) demonstrated

synthesis of IAA in cultures of *Rhizopus suinus* and more recently Brian (1957) examined twenty-five species of fungi for IAA production. He included in his study fungi from different taxonomic groups and concluded that none of them produce more than a trace of IAA in media of glucose and salts, but most of them produced some, if tryptophane was supplied as a precursor. The production of IAA *in vitro* is inhibited by the production of extra-cellular IAA-destroying enzyme and this limits the quantity of IAA produced. IAA is considered to play a major role in cell division and extension and therefore IAA production by parasites leading to excessive IAA levels *in vivo* has been thought of as a possible cause of hypertrophy or hyperplasia accompanying certain plant diseases. As emphasised by Brian (1957) this implies that the parasite transforms host tryptophane to IAA better than the host enzymes and also that the host IAA oxidases which help to keep down IAA below certain levels in healthy tissues do not help in removal of the extra IAA produced by the parasite.

In the case of maize smut caused by *Ustilago maydis,* large conspicuous globose galls are produced on the leaves, stems and inflorescences. These galls have much more auxin than is found in healthy tissues of maize. *U. maydis* is known to produce IAA *in vitro* (Moulton, 1942; Wolf, 1952) and there is a direct correlation between the capacity of various strains of this pathogen to produce auxin *in vitro* and their pathogenicity. Another interesting fact is that a different smut, *Ustilago nigra* which infects barley incites no galls in the host tissues; nor does it produce any IAA *in vitro.* In the case of infections by rusts also, abnormally high IAA content has been recorded for infected tissues showing proliferation. Hirata (1954) studied the auxin content of healthy tissues and tissues infected by different rusts, using the *Avena* coleoptile test. His investigations with aecial stages of *Gymnosporangium haraeanum* on *Pyrus montana* v. *rehderi, Puccinia caricis blepharicarpae* on *Smilax china,* and *Uromyces alopecuri* on *Ranunculus vernyii* v. *glabe* indicated 1·6, 5·0 and 13·4 times as much auxin, respectively, in diseased as in comparable healthy tissues. Theoretically, the increase in auxin may be through direct synthesis by the parasite, or by the host, or the result of host-parasite interaction. The successful culture of *Gymnosporangium juniperi-virginianae* by Hotson & Cutter (1951) paved the way for further work on this problem. For instance, Wolf (1956) has recently reported that IAA is produced as a metabolic product during growth *in vitro* by *Gymnosporangium juniperi-virgini-*

anae. The quantities of IAA produced by the parasite were $2{\cdot}2 \times 10^{-5}$M after 10 days' incubation, 4×10^{-5}M after 17 days, and $1{\cdot}1 \times 10^{-4}$M after 24 days. Wolf also reported on the pathways leading to IAA production; by chromatographic techniques it was shown that the formation of IAA from its precursor tryptophane involves tryptamine and indoleacetaldehyde as intermediates. Mention may be made here also of invasion of apple stems by *Nectria galligena* which results in an intensive proliferation of cells surrounding the point of entry. In this case, again, IAA has been shown to be produced *in vitro* by the fungus (Berducou, 1949). On the other hand, another fungus of the same genus, *N. corallina* which also infects fruit trees, hardly causes any proliferation of host tissues and does not produce *in vitro* any IAA either. The examples just cited have been considered to point to a definite role for IAA in the genesis of the disease syndrome typical of the hyperplastic diseases.

Crown gall which is caused by *Agrobacterium tumefaciens* has been studied intensively by a number of workers. Three major periods of change are reported to be involved in the genesis of the crown gall tumour from normal cells (Klein, 1954) : (i) the transformation period which includes pre-induction, induction, promotion and completion, and the end result of which is a primary tumour cell; (ii) the duplication period during which the primary tumour cells and their progeny undergo a series of rapid divisions and during which secondary transformation processes may also function; these divisions result in an undifferentiated tumour mass; and (iii) the organization and differentiation period during which structural differentiation of the tumour mass occurs. A characteristic internal organization, the ability to form teratoma shoots, and possession of distinct physiological and biochemical mechanisms typify the mature crown gall tumour. During the transformation period, wound substances released from the wounded cells act on normal cells and alter them to *conditioned cells,* but the bacteria are not required for these changes. This is followed by the induction phase when conditioned cells are acted upon by tumour inducing principle (TIP) which is formed by virulent bacteria with the proper hereditary constitution in the presence of wound substances. It is believed that this principle may be a specific deoxyribonucleic acid (DNA) and its action on conditioned cells transforms them into *incipient tumour cells.* According to Klein (1954) auxin would appear to be the only substance acting on incipient tumour cells and

transform them into enlarged *promoted cells,* the auxin possibly being synthesized by virulent bacteria or being produced by the incipient tumour cells themselves or by bordering tissues.

Following bacterial action, the affected host cells acquire the ability for continued and rapid unregulated proliferation which is entirely independent of the bacteria. In 1942, White & Braun showed that when fragments of bacteria-free tumour tissues were placed in a healthy host of the same species, the fragments developed into typical crown gall tumours resembling those originally initiated by the bacteria (see Braun, 1954). Thus, it would appear that some factor passes from the bacteria into the host cells which influences the subsequent behaviour of these cells. There is considerable evidence suggesting that, as a result of their transformation, crown gall tumour cells become endowed with a capacity to produce growth substance in excessive amounts and this may be the cause of the unregulated proliferation. How is this growth substance imbalance brought about? At least three different hypotheses have been advanced to explain this. For instance, the observation that this capacity for autonomous growth arises suddenly in a cell and becomes a fixed character transmitted indefinitely to daughter cells is considered to indicate that the change involves a somatic mutation resulting in modification or destruction of one or more genes in tumour cells. A second possibility is that the growth substance imbalance might be due to the presence in these cells of a new self-duplicating agent such as a virus that is transmitted by bacteria. The virus hypothesis was advocated by de Ropp in 1947 (see Braun, 1954); he found that when bacteria-free tumour tissue scions were grafted on to normal sunflower stocks, secondary tumours were produced. The discovery of a new protein of high molecular weight in crown gall tissue and its absence in normal tissue is interesting in this connection. However, no one appears so far to have demonstrated virus particles in crown gall tissue with the aid of the electron microscope. Braun (1954) has recently outlined the arguments against this view. The third hypothesis put forward to account for the growth substance imbalance is that one of the normal cytoplasmic systems may have been modified as a result of bacterial action.

Braun's (1957) more recent work has thrown further light on problems relating to tumour formation in crown gall. As already mentioned, the tumour cell is an altered, randomly proliferating

cell that has become more or less independent of the morphogenetic restraints that govern the growth of normal cells within a living organism. It shows the capacity for autonomous growth. Whilst it must be conceded that tumour genesis may result from the effects of different factors such as radiant energy, chemicals, microorganisms, viruses, etc., it is yet possible that these factors affect a common cellular mechanism or mechanisms, which when once deranged, continues to stimulate abnormal proliferation of cells. Such proliferation is the result of the twin processes of cell enlargement and cell division. Specific substances synthesized in plant cells are involved in both these processes. It is now known that only when an auxin and a cell-division factor are both present would profuse growth accompanied by cell division take place. In the case of crown gall tumour tissue evidence of presence of both cell enlargement and cell division factors is available (see Braun, 1957). In the transformation of a normal cell into a tumour cell, a permanent activation of these two growth-substance-synthesizing systems takes place leading to quantities of the cell enlargement and cell division factors far in excess of what is normal, which eventually bring about continued and abnormal proliferation. Braun's (1957) work has further shown that there may also be an activation of additional growth substance systems involved in the transformation of the normal cell to a tumour cell. The alteration of normal cells to tumour cells is a gradual process. When the bacteria are allowed to act for a 3-4 day period on the normal cells, rapidly growing potentially malignant tumour cells are formed. However, if the bacteria are allowed to act only for a shorter period, (say, 34-36 hours) [this would be possible by selective killing of bacteria by thermal treatment], slow-growing, somewhat benign tumours are produced. The characteristic growth patterns of these two types of tumour cells are maintained indefinitely *in vitro* and therefore it would appear that the extent of cellular alteration attained in a 34-36 hour period is much less than that found when the bacteria act on host cells for a period of three days. It is thus obvious that in a 34-36 hour period only a partial cellular alteration leading to a low grade of neoplastic transformation is effected. From a critical study of tissues that have undergone such partial alteration, it has been demonstrated that when these tissues were planted on a basic White's medium supplemented with naphthalene acetic acid (NAA) [which induces cell enlargement] or with a cell division factor, the growth rate of these tissues was not greater than

when they were planted on the basic medium without these supplements. Even a combination of an auxin and a cell division factor did not stimulate increased growth of these tissues and it is therefore clear that neither of these two factors, alone or in combination, were limiting factors for autonomous growth of these tissues. On the other hand, when 1·0 mg./ml. of yeast extract and 0·1 mg./l. of NAA were added to the basic medium, the rate of growth of cells altered in 34-36 hours was comparable to that of cells altered in a 3-4 day period; yeast extract or NAA added singly were ineffective. Thus, it would appear that yeast extract includes some essential factor(s) for growth. Further, the capacity of these cells for synthesizing auxin was permanently fixed at the time of their conversion to tumour cells at a level suboptimal for rapid autonomous growth. The ability of these tissues to proliferate in the presence of added NAA and yeast extract is lost by repeated transfer to the basic medium and is therefore not an example of acquired capacity for growth (Braun, 1957). Addition of optimal levels of NAA and suboptimal levels (1/10th of optimum) of yeast extract to the basic medium was ineffective, but the further addition of acid hydrolyzed casein or of pure amino acids used in the proportions found in casein hydrolysate were effective. It would be safe to assume that the transformation of a host cell into a tumour cell leads to a permanent activation of several quite distinct metabolic systems. "Whether cellular systems controlling the availability of cell enlargement, cell division, amino acid and yeast extract factor or factors are somehow coupled and therefore fixed simultaneously by the tumour-inducing principle, or whether they represent entirely different systems that are acted upon independently by the tumour-inducing principle, or whether, in fact, there is some master reaction within the cell that is specifically affected by the tumour-inducing principle, thus accomplishing the simultaneous activation of all growth-substance-synthesizing systems cannot be determined from the information now available". (Braun, 1957, p. 140-41).

LENGTHENING OF STEM

We may now pass on to examine briefly examples where a conspicuous lengthening of the stem forms part of the disease syndrome. In the case of infection of *Sempervivum* spp. by the rust *Endophyllum sempervivi*, and of *Euphorbia cyparissias* by the rust *Uromyces pisi*, lengthening of stems has been reported. In the case of the former, Pilet (1952) assayed chloroform extracts

of leaves for auxin by the *Avena* coleoptile test and found that normal leaves had 10^{-7}M IAA, whereas the IAA content of the diseased leaves in the vicinity of the lesions was 10^{-4}M. Studying infection of *Euphorbia* by the aecidial stage of *Uromyces pisi* in the same way, Pilet (1953) found that the IAA content of diseased leaves was 10^{-4}M, whereas that of healthy leaves was only 10^{-6}M. Thus, in these two cases of infections by obligate parasites, there was a possible correlation between IAA production and stem lengthening. On the basis of these results, Pilet enumerated three possibilities: (i) the parasite produces IAA; (ii) IAA is produced by the host as a reaction to the presence of the parasite; and (iii) the parasite produces a precursor of IAA which is modified in the host tissues. However, when externally applied, IAA does not normally induce any considerable lengthening of stems (see Brian, 1957). As has been pointed out by Brian (1957), the increased IAA in diseased tissues is perhaps only incidental and not causal in these cases. The same criticism applies also to the more recent work of Daly & Inman (1958) who have suggested a correlation between elongation of safflower plants infected by *Puccinia carthami* and their auxin content. They were able to detect more IAA in extracts from rust-infected than in healthy tissues.

Another well-known example where stem lengthening is a characteristic symptom is the bakanae disease of paddy which is caused by *Gibberella fujikuroi*. Elongation of internodes of diseased plants results in plants abnormally taller than healthy ones. The pioneering work of Kurosawa indicated that similar symptoms could be produced in paddy seedlings treated with cell-free culture filtrates of the pathogen. Some years later Yabuta & Hayashi (see Brian, 1957) isolated a crystalline material from culture filtrates of the fungus which they called gibberellin A (formula $C_{22}H_{26}O_7$) and which was shown to produce lengthening of stems not only of paddy plants but also several other monocots and dicots. More recently, another metabolic product, viz., gibberellic acid ($C_{19}H_{22}O_6$) has been isolated from the same pathogen and has been shown to have similar physiological effects on plants (see Brian, 1957). On paddy plants, gibberellic acid (GA) brings about symptoms typical of the bakanae disease, i.e., lengthening of internodes and leaves, slight chlorosis and reduced tillering. The demonstration of the production of GA *in vivo* within plants naturally infected by *Gibberella fujikuroi* (Subba-Rao, 1957b) indicates that the symptom picture in the bakanae disease is due, at

least in part, to GA produced *in vivo*. Considering, in general, the physiological effects of GA on living cells, it may be summarised that in the majority of cases, the most notable effect is cell extension. However, a few possible exceptions have also been reported. For instance, in the case of rosette plants of *Hyoscyamus,* GA stimulates lengthening of stem and since in this case internodes are practically non-existent, it has been suggested that additional cell division should precede cell elongation (Lang, 1956). Working on the rosette plant *Lactuca scariola,* a similar effect was described by Lona (see Brian, 1957).

EPILOGUE

In the two talks I have given, I have mainly dealt with some aspects of the mechanisms underlying certain distinct symptom pictures, such as wilting, necrosis, hyperplasia, etc., which characterise some diseases of plants. The treatment of the subject is not exhaustive, but some current trends of thought have been outlined. A plant physiologist would have taken up the different life functions of a plant such as its water relations, carbohydrate and protein metabolism, etc., and discussed how these are deranged following infection and disease development; but, as a plant pathologist, I have viewed the subject from a different angle and I have tried to explain in a general way the *modus operandi* of pathogens in producing the distinctive and characteristic symptoms on their respective hosts.

In what I have covered, I have tried to illustrate the points I wanted to emphasize with selected examples. For this reason I should caution you not to tend towards too much of generalisation. As I said in the beginning, it will be difficult to generalise for two reasons: firstly, because we do need more data on many aspects of physiology of disease; secondly, because the subject of physiology deals with functional mechanisms, and the living cell, being what it is, eludes the comprehension of even the most conscientious, industrious and tenacious worker. Anyone who has worked on physiological problems will concur with me here. All the problems which I referred to yesterday and today need more intensive study with well-defined material. Such studies will also have to be extended to a variety of material later. Intensive study, in my humble view, should precede any broad-based approach to problems in physiology. I say this mainly for the younger workers in the audience before me. Another point I

wish to make here is one which has all along been so dear to my teacher, viz., the need to devise new techniques, be they physical, biochemical or otherwise, and to improve older ones. No one working on living plants and trying to understand their life processes can afford to ignore this. There is also a very great need for workers to come together and put in their efforts as a team. The whole problem of deranged physiology of the diseased plant has great scope for future work and I do hope this subject will attract greater attention from workers in this country.

My main aim in speaking on the subject has been to enthuse younger workers to take up for study problems in physiological plant pathology seriously and, if I have succeeded in having done this, I should be more than pleased and satisfied. In any case, I certainly have the satisfaction of having learnt a good deal in having tried to arrange my thoughts for these talks.

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A Note on *Euphorbia Longistyla* Boiss

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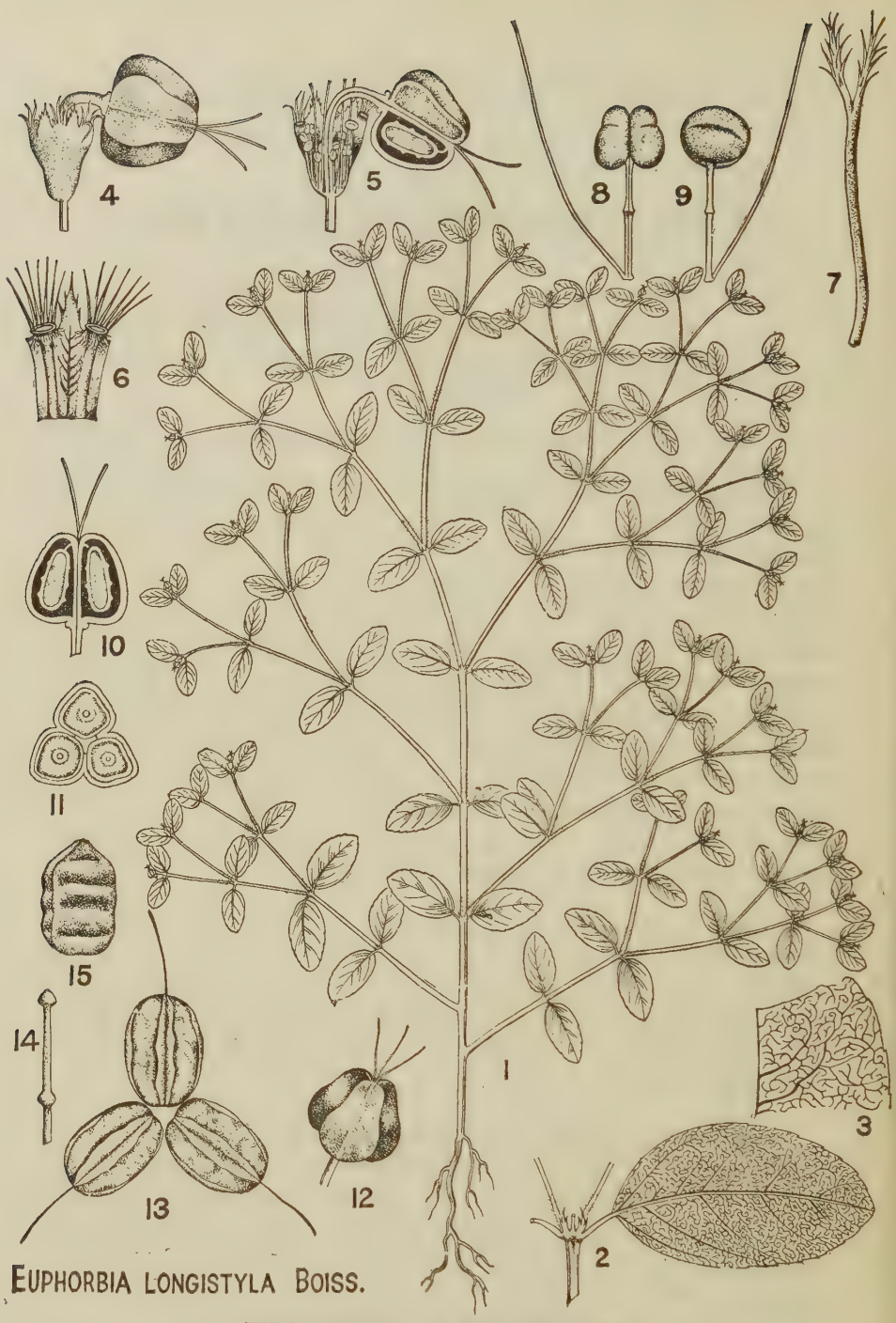
During a botanical exploration in the Cuddapah District, Andhra Pradesh, in October 1958 (19th to 24th) a population of *Euphorbia longistyla* Boiss. was noticed at Guvvalcheruvu, Palkonda Reserve Forest, at an altitude of 700 meters. This plant is neither represented in the Madras Herbarium nor is there its diagram in Wight's Icones (1838-53). Further Hooker (1885) remarks on this plant: "I have seen only very imperfect specimens of this plant in Wallich's Herbarium and have taken its characters chiefly from Boisser, who says that it probably assumes a form with imbricate floral leaves". In view of the imperfect data on this plant, extensive collections were made and 300 specimens were carefully examined. The present note relates to a detailed taxonomic description of this plant (Figs. 1-15).

The distinguishing characters of *E. longistyla* are: herbaceous dichotomous habit with filiform end-branchlets; opposite, oblong-elliptic, oblique, membranous leaves; smaller floral leaves; terminal involucres; multifid limb of glands; three conspicuous, long, filiform styles; glabrous, trigonous, keeled capsule; and transversely furrowed, tetragonous seeds.

Herbaceous, annual, erect, much-branched, nearly 35 cm. high; stem woody, dichotomously branching, yellowish-brown, almost glabrous, base of branches velutinous, ending branchlets filiform; leaves opposite, simple, elliptic-oblong, oblique, obtuse, membranous, minutely serrulate towards the apex, 8-22 mm. long, 4-12 mm. wide; petioles 0·75-2·25 mm. long; stipules minute, lanceolate-acuminate, incised, scaly; involucre terminal, turbinate, glabrous, 1·25-2·25 mm. long, 0·75-1·5 mm. wide; peduncles 1·25-3·5 mm. long; lobes 5, each multifid, central lobe longest, minutely tuberculate, margins velutinous; glands 4, capitate, ovate with a



EXPLANATION OF TEXT-FIGURES

FIG. 1. Habit (Half nat. size). FIG. 2. Node enlarged showing vegetative parts x 2. FIG. 3. Leaf venation enlarged x 4. FIG. 4. Cyathium x 5. FIG. 5. L. S. Cyathium x 5. FIG. 6. Involucral glands with multified limbs x 10. FIG. 7. Peripheral bracteole x 10. FIGS. 8 and 9. Male flowers with bracteoles x 10. FIG. 10. L. S. Ovary x 5. FIG. 11. T. S. Ovary x 5. FIG. 12. Fruit x 5. FIG. 13. Cocci x 5. FIG. 14. Columella x 5. FIG. 15. Seed x 10.

distinct rim, shortly stalked, very velutinous around the base of its stalk, arranged in the sinuses of involucral lobes, leaving one sinus empty; limb of glands multifid, cleft to the base into 4-7 linear, minutely tuberculate bristles, 0·5-1·25 mm. long, pink; male flowers in 5 fascicles, each fascicle 5-7 flowered, supported by filiform bracteoles protruding out of the cup, the peripheral bracteoles adnate to the involucre with lacinate tips; male flowers naked, articulate over the pedicel; anthers sub-globose, dehiscence transverse; stalk of ovary 1·75-2·75 mm. long, recurved in fruit, glabrous, styles 3, filiform, pinkish-red, free to the base, microscopically tuberculate at the base; stigma simple; fruit 2-2·75 mm. long, 2-3·5 mm. wide, dehiscing into three 1-seeded, 2-valved cocci, leaving a persistant central columella, cocci glabrous, keeled; seed obtusely four angled, transversely 3-4 furrowed, 1-1·5 mm. long, 0·75-1·0 mm. wide, pinkish-red in colour.

The natural distributional area so far known for *Euphorbia longistyla* is the Ceded Districts (Andhra Pradesh) upto 700 meters.

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Holotrichous Ciliates from Madras

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Introduction

The freshwater ciliates occurring in India have been studied by several workers, and the first comprehensive treatise on Indian ciliates was published by Bhatia in 1936 in the Fauna of British India series. This monograph, incorporating largely the work of various authors however deals only with freshwater and parasitic forms. Khajuria (1950) has recorded a few species of ciliates from Benares Cantt. Except for the work of Ganapathi and Narasimha Rao (1958) who have recorded marine ciliates from Visakhapatinam, there has been no published account of ciliates from marine localities. In the present paper, Holotrichous ciliates collected from inshore plankton and brackish waters, along with the bottom mud are described. Kahl's (1930) system of classification has been followed.

Material and Methods

The marine forms described here were picked off from samples of inshore plankton brought to the laboratory, while others were collected from samples of water and bottom sediments from the Adyar and Cooum estuaries. In some cases the numbers collected were increased by culturing them in finger-bowls in the laboratory. Sodium amytal, methyl cellulose, weak iron-alum solution or osmic acid vapour were used to immobilise these rapidly moving ciliates. Study of the ciliates in the live condition was facilitated by the use of highly diluted solutions of Congo red, methylene blue or neutral red which stained organellae and inclusions without injury to the rest of the organism. Permanent preparations were made first by fixation in hot Schaudinn's fluid or weak Flemming's followed by staining in Heidenhain's iron-haematoxylin or Delafield's haematoxylin. Details of nuclear structure were revealed clearly by the Feulgen's technique.

List of the Species

1. Family *Holophryidae*
   Genus *Holophyra*
   Holophrya indica
   Genus *Urotricha*
   Urotricha globosa.
   Genus *Lacrymaria*
   Lacrymaria olor.
2. Family *Trachelocercidae*
   Genus *Trachelocerca*
   Trachelocerca phoenicopterus.
3. Family *Colepidae*
   Genus *Coleps*
   Coleps hirtus.

Description of the Species

Family *Holophriydae*
Genus *Holophrya*

1. *Holophrya indica* Bhatia, 1916
   Holophry indica Kahl, 1930

The body is slightly flexible and elliptical with both ends rounded. The cuticular surface presents distinct alternating longitudinal striae and furrows. The body is ciliated uniformly throughout, the cilia being long and disposed along the longitudinal striae.

The mouth is situated at the anterior pole, with the border not projecting out. A pharynx is absent. The macronucleus is large, horse-shoe shaped and situated in the anterior half of the body. The contractile vacuole is single, spherical and posteroterminal with a cluster of small circular feeding vacuoles in its neighbourhood. (Fig. 7).

Collected from the Cooum estuary.
Size : Length—100μ.
Breadth—50-60μ.

Genus *Urotricha*

2. *Urotricha globosa* Schewiakoff, 1889
   Urotricha globosa Bhatia, 1916

The body is elliptical. The circular oral aperture is at the anterior end, which is more pointed. The mouth is slightly prominent and leads into a large pharynx. One large contractile vacuole is present in the median line, of the posterior half of the body. The macronucleus is large spherical and placed almost at the centre of the body with a small micronucleus lying near it. The body surface is entirely ciliate. The cilia near the mouth are longer, while those towards the posterior end are slightly shorter. The cilia vibrate independently and irregularly. From the posterior end springs one single seta or springing bristle, elongated in the long axis of the body. Longitudinal striations may be observed along the body surface running from end to end.

These organisms were recorded from the interstitial habitat of the sandy beach. (Fig. 4).

Size : Length—15-20μ.
Breadth—12-15μ.

Remarks :

The present marine form *U. globosa* agrees in it's description with that given by Bhatia of a freshwater form recorded at Lahore, in the general body outline, longitudinal striation of the body surface, the swift movement, the direction of which is often changed and in the possession of a springing bristle at the posterior tip of the body. In the present form and that recorded by Bhatia the macronucleus is spherical and proportionately larger than that figured by Roux for the type. The contractile vacuole is in the median line near the posterior part of the body and not on one side. In the nature of the contractile vacuole and the macronucleus this form resembles that of Schewiakoff. Khajuria (1950) has described a fresh water species of *Urotricha* from Benares as *U. bhatiai,* which he considers different from *U globosa.* In spite of the fact that the present form has cilia on either side of the springing bristle, as in *U. bhatiai,* the present author considers the Madras form different from *U. bhatiai* in (a) the body form being distinctly elliptical, both sides being convex as in *U. globosa* (Schewiakoff). (b) the food-vacuoles which are not so few or large as figured by Khajuria. (c) the springing bristle in *U. bhatiai* is longer than the body while in the Madras form it is much shorter. However the present author feels that the above mentioned points of difference do not justify the creation

of a new species though Khajuria is of opinion that uniform ciliation and a few large vacuoles are of "sufficient specific value" to justify the creation of his new species.

Genus. *Lacrymaria*

3.	*Lacrymaria olor*	O. F. Müller, 1786
	Vibrio olor	O. F. Müller, 1786
	Lacrymaria olor	Kahl, 1930

The anterior half of this ciliate is long and narrow, extremely contractile and slightly flattened in the dorso-ventral plane, while the posterior half is more rounded and cylindrical, resembling the base of a flask, but with a slightly rounded or pointed tip. At the anterior extremity is the well-developed oral come. However there is no distinct furrow between the oral cone and the rest of the body as is seen in most species of the genus. The cytostome is small and slit-like, leading into a pharynx which is not very well developed. A circlet of slightly longer cilia is present round the base of the oral-cone. The body exhibits light striations on the surface. Cilia are arranged along the entire body surface in spirals, well seen on the posterior half of the body. The macronucles is large and bipartite, with a constriction in the middle, near which lies the micronucleus. Two contractile vacuoles are present, one anteriorly near the junction of the neck with the base and one at the posterior end of the body.

The neck-like part of the ciliate is capable of considerable extension and retraction as well as lateral movement. (Figs. 1, 3, 5).

Collected from a sample of water and soil from the Cooum estuary.

Size: The length of the animal varies from about 100-450 μ according to the extension of the neck.

4. *Lacrymaria coronata* : Claparede & Lachmann, 1858.

The body is elongate, clavate and cylindrical, slightly narrowing anteriorly. The absence of a neck which is a conspicuous feature of the previous species, distinguishes the present form. Body moderately elongate but not capable of such extreme contraction and expansion as in *L. olor*. The body is divided

distinctly into two parts the anterior being conical and short. The posterior part is extremely long, almost uniformly cylindrical and slightly pointed at the posterior end. The apical end or anterior region is separated from the rest of the body by a circlet of cilia, lying within a furrow or grove. At the apical extremity occurs the mouth, leading into a pharynx.

The elongated posterior portion of the body is entirely covered with fine cilia, arranged in oblique spirals Contractile vacuole is single, spherical and postero-terminal. The macro-nucleus is elongated band-like and curved.

Most often the ciliate moves in a gliding manner but at times it spins and progresses spirally. (Figs. 2, 6, 11).

Collected from the Cooum estuary.

Size: Length 120-150 μ.
Breadth 35-40 μ.

Genus. *Trachelocerca*

5. *Trachelocerca phoenicopterus* — Cohn, 1856
Trachelocerca fasciolata — Sauerbrey, 1928

The body of the animalcule is highly elongated and ribbon-like, retractile and elastic. The anterior end of the body is slightly wider and prolonged into a transparent neck. The posterior end forms a long pointed tail-like portion Ganapathi and Narasimha Rao (1958) report of tail-less forms from Visakhapatinam and Lebedew (1909) is of opinion that the tailed and tail-less forms represent two varieties. The oral aperture is terminal with a rigid and annular border, followed by a funnel-shaped pharyngeal cleft.

The body is finely ciliated throughout along longitudinal rows. The tail is drawn out into a long pointed tip and the cilia on this elongation are slightly longer than those of the general body surface. The tail can often be seen anchoring the animal to bits of debris while the rest of the body is moved in various directions. The body presents annular striations which cannot be seen in a state of contraction.

The macronucleus as recorded by Entz (1884) and Butschlii (1881), consists of a single round body, while Kent (1881) describes it as consisting of two parts and Gruber, Kahl and Calkins

(1901) as consisting of many parts scattered along the length of the body. Kudo (1954) restricts the number to four radially arranged endosomes. In the present form, the macronuclei are seen to be many scattered along the length of the body. The micronuclei have not been observed. The contractile vacuoles are multiple and lineally disposed, though they are not clearly visible. (Figs. 8-10).

Collected from the Cooum estuary along with the soil.

Size : 1. m.m. — 1·5 m.m.

Genus *Coleps*

6. *Coleps hirtus* : O. F. Müller, 1786

The body is elongated along the antero-posterior axis, almost twice as long as broad. The anterior margin of the body is denticulate and provided with a circlet of oral cilia, which are longer than those of the body. The cuticular surface of the body is marked by distinct transverse and longitudinal furrows, into a number of quandrangular facets. These facets are smooth and indurated and the narrow furrows intervening between the facets are provided with cilia. The mouth is at the anterior end surrounded by the oral circlet of cilia. A distinct transverse furrow divides the body into two equal anterior and posterior halves. The macronucleus is large and rounded and placed in the anterior half of the body, with the micronucleus lying near it. The contractile vacuole is single and posterorly situated. The posterior end of the body is rounded and provided with three spines and a caudal style.

Locomotion is very rapid. The organism keeps rolling and revolving with sharp and swift changes in the direction of locomotion. (Fig. 12).

Collected from the Cooum estuary.

Size : Length 40-70 μ.
Breadth 20-30 μ.

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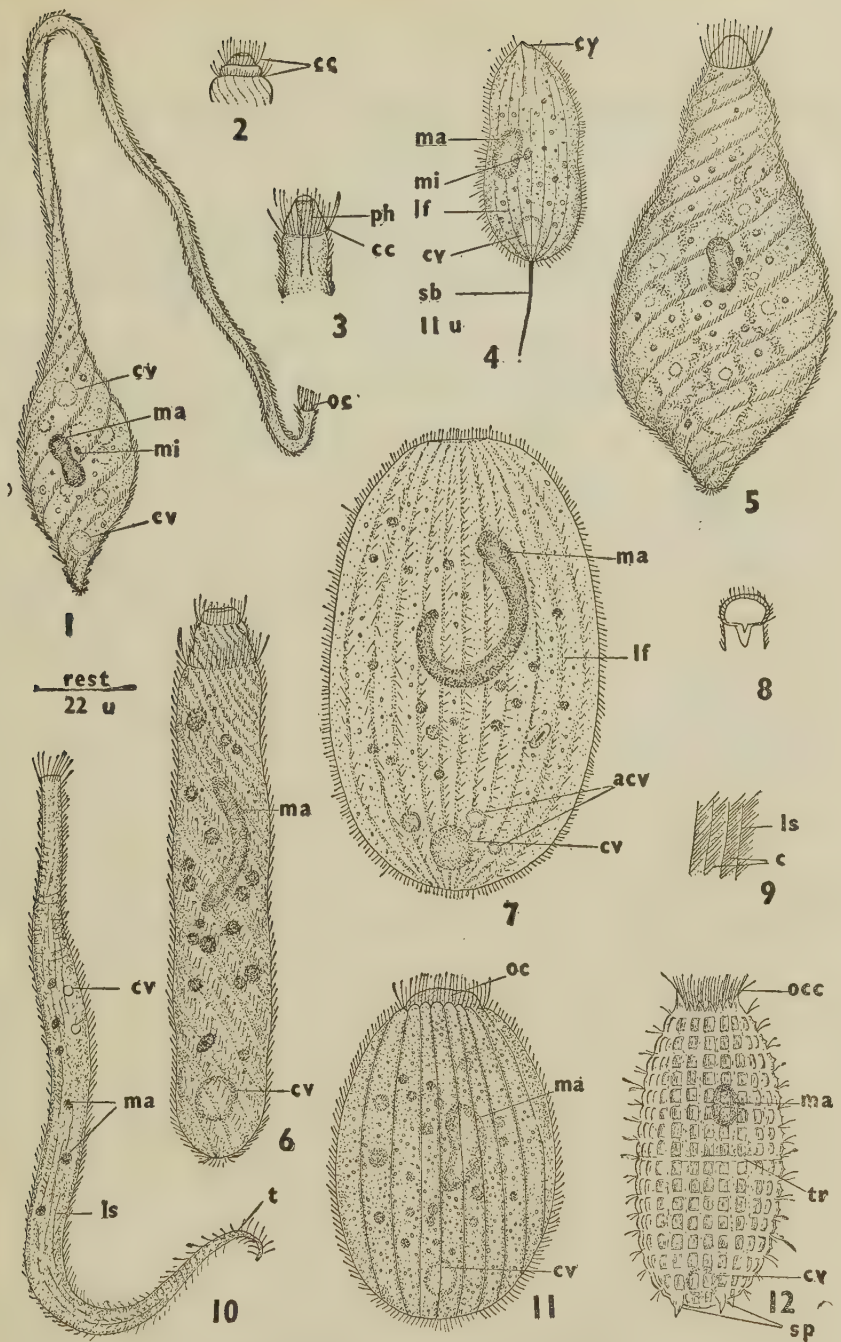

EXPLANATION OF TEXT-FIGURES

Figs. 1, 3, 5. *Lacrymaria olor,* 1 in extended state, 3. anterior and enlarged and 5. in contracted state; Figs. 2, 6, 11. *L. coronata* 2. anterior end enlarged, 6. in extended state and 11. in contracted state; Fig. 4. *Urotricha globosa;* Fig. 7. *Holophrya indica;* Figs. 8-10. *Trachelocerca phoenicopterus,* 8. anterior end enlarged, 9. a portion of the body enlarged showing ciliary arrangement; Fig. 12. *Coleps hirtus:* (*acv*—accessory vacuoless; *c*—cilia; *cc*—ciliary circlet; *cv*—contractile vacuole; *cy*—cytostome; *lf*—longitudinal furrows; *ls*—longitudinal striations; *ma*—macronucleus; *mi*—micronucleus; *oc*—oral cone; *occ*—oral circlet of cilia; *ph*—pharynx; *sb*—springing bristle; *sp*—spines; *t*—tail; *tr*—transverse furrow).

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A Case for Seed Certification and for Organising Crop Improvement Associations in India[1]

By

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The importance of pure seed in the improvement of crops cannot be over emphasized. It is not uncommon that the cultivators get greatly disappointed and realise rather too late for not bestowing proper attention to the seed at the time of sowing. The low quality seed will result in either weak plants which very often wither away leaving uncovered areas in the field or owing to the poor viability of the seed there may be failure of germination resulting in the loss of the season. The crop also may not come to harvest at the same time. Some plants flower early and shed their seed long before harvest while others may still remain unripe at the time of harvest. This results in considerable loss to the cultivators. By using good seed alone and eliminating these defects, it may be possible to increase the yields upto ten percent or more. Further, the use of impure seed has the lurking danger of introducing unwanted plants which may later turn out to be the worst pernicious weeds. A good quality seed must be pure without any admixture of impurities, true to its heredity and should possess a high percentage of viability. The fundamental purpose of seed certification is, therefore, to make available to the grower good quality seed of known purity and heredity.

Production of pure seed in India

In India there are no recognised seed growers of agricultural crops except for a few seedsmen who deal mostly in horticultural crops and vegetables. The responsibility for the production of improved seeds of agricultural crops is now entirely with State

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Departments of Agriculture. The nucleus seed is produced on the Agricultural Research Stations and this is multiplied in two or three stages on cultivator's field under the supervision of the departmental officers and distributed to the cultivators through the depots of the Agriculture Department. No doubt the scheme is good, however, it lacks the element of competition and comparison which are essential for maintaining the quality of the seed. Moreover in this scheme, the producing as well as the certifying agencies are one and the same which can with advantage be avoided. Further the state enterprise involves a huge overhead expenditure which is a perennial drain on the States' exchequer. Therefore, it may be desirable to encourage private seed trade in this country as it now exists in the advanced countries like the United States of America.

A knowledge of the methods of seed multiplication and distribution as exists in the United States of America may be helpful in this regard. The author had the privilege of studying these processes in the States of Mississippi, Minnesota, Illinois and Nebraska. A brief outline of the working of the seed multiplication and distribution programme in that country is given below :

Seed Laws

The seed multiplication and distribution in the United States of America are governed by both the Federal seed laws and State seed laws which set out regulations relating to the quality, sale, transportation and distribution of seeds. Agricultural and vegetable seeds imported into the United States of America must meet certain minimum standards for pure live seeds, must not contain more than two per cent by weight of weed seeds and must not contain the designated noxious weed seeds. Agricultural seed moving in interstate commerce is required to be truthfully labelled to show :

i. The name of each kind or variety.
ii. Percentage of pure seed.
iii. Other crop seeds.
iv. Weed seeds.
v. Inert matter.
vi. Germination and hard seed when present.
vii. The name and rate of occurrence of permissible noxious weed seeds.
viii. The name and address of the shipper.

There is considerable variation in detail among the seed laws from state to state: for they differ in the noxious weed seed requirements and also each state has its individual list of noxious weed seeds.

In order to carry out the intent of the laws pertaining to the seeds each State or the Federal agency charged with the administering such laws must have the means of testing seed samples. The seed laws in most States are administered by the State Departments of Agriculture although in a few States administration of such laws is the responsibility of the State Colleges of Agriculture or Experimental Stations. The Federal Seed Act is administered by the Grain Branch Production and Marketing Administration, United States Department of Agriculture.

Seed Certification

The purpose of seed certification is to make available to the grower high quality seeds and propagating materials of superior crop plant varieties so grown and distributed as to maintain high quality genetic identity and purity and thereby giving the purchaser an honest and unbiassed opinion of the merit of such seed.

The seed certification is done by the Crop Improvement Association in each State which is recognised as the official agency for certifying the seed. To give an idea of the constitution of the Crop Improvement Association, some of the features of the Association in the State of Mississippi are outlined below.

Crop Improvement Associations

The Crop Improvement Association is a Farmers' Co-operative Organisation operating under an Act of the State. This Association is recognised by the State law as the official seed certifying agency.

The Board of Directors of the Crop Improvement Association is composed of seven members. The Board is elected by and from the membership. The terms of office of the Board members are staggered in such a way that not more than two members are retired in any one year.

The Seed Certification Committee consists of the President and Vice-President of the Association and one other member of the Board of Directors appointed by the President.

The Association works in close co-operation with the Experiment Station personnel on technical matters pertaining to various crops and seeds. Whenever questions arise pertaining to a certain crop, the Experiment Station Specialists directly concerned with this crop are consulted and asked for advice and recommendations.

The Association adopts the various standards for certification prescribed by the State Department of Agriculture for each crop. The State Seed Laboratory, under the State Department of Agriculture, makes the official seed analyses for the Crop Improvement Association.

Finances for the Crop Improvement Association are obtained through the field inspection fees and fees for tags and seals.

The Executive Secretary of the Association who is generally a graduate of Agriculture is either selected by the Association or appointed by the State Department of Agriculture. The other personnel of the Association are appointed by the Executive Secretary. The Association has one or two Field Inspectors who are also graduates in Agriculture.

The association has the following objectives:

i. Supply each grower with instructions and material for making application for field inspection.
ii. Make field inspection of all the crops at proper time.
iii. Take official samples from seed lots processed for sale and make laboratory tests.
iv. Issue certificates, certification tags and seals for seed lots that qualify for certification.
v. Publish directories of registered and certified seed for the benefits of the growers.

Seed Multiplication

The seed trade in the United States of America is entirely in the hands of the private seed growers. As per the seed laws, the seed that is put for sale should meet with certain minimum standards. The growers usually get their seed officially certified by the Crop Improvement Association in each State. Each certified grower must take out a licence for growing a particular seed subject to the following conditions:

i. He should make out an application to the Crop Improvement Association for certification and pay the prescribed

fees for field inspection, official sampling and laboratory tests.

ii. He must show evidence of good management of the farm and evidence that reasonable precautions are taken to control the seed borne diseases.

iii. He must have enough land to satisfy the requirements of isolation standards.

iv. He must rogue the fields periodically and remove all undesirable plants, like off-type plants, prohibited and restricted weeds, other crop plants etc., which may be growing in the field. In hybrid seed corn production, off-type plants should be removed before the pollen is shed.

Classes of Seed

There are generally three classes of seed as detailed below:

(i) *Foundation seed*: Foundation seed constitutes the traceable progeny of a variety or strain produced by individual plant, head or pod selection or mass selection of plants, heads or pods or by controlled self pollination and cross pollination of recorded origin in the hands of the original breeder or experiment station.

(ii) *Registered Seed*: Registered seed is the progeny of the foundation seed that is so handled as to maintain the satisfactory genetic identity and purity and that has been approved and certified by the certifying agencies.

(iii) *Certified Seed*: Certified seed is the progeny of the registered seed that is so handled as to maintain the satisfactory genetic identity and purity and that has been approved and certified by the certifying agencies.

When there is great demand for a certain variety and the available registered seed is limited, it is usual to multiply the certified seed and obtain a second generation certified seed.

The seed bags containing three classes of seed are given differently coloured labels so that a buyer can at sight make out the class of seed he is buying without going through the contents of the tag.

Foundation seed is produced generally on the experimental station under the supervision of the State Plant Breeders and is distributed to the registered seed growers. Producers of the registered seed are selected primarily on the basis of a satisfactory

record of certified seed production. The production of the certified seed is open to any one who agrees to comply with the rules and regulations established by the Crop Improvement Association detailed above. Growers with a satisfactory record of at least one year of certification will be given preference.

Field inspection

When a certified seed grower takes out a licence for producing the seeds, his fields are periodically inspected by trained Inspectors employed by the Crop Improvement Association. Field inspections are done at least twice during the period of the crop viz., once during flowering and the second prior to harvest. The Inspector makes a report on the condition of the crop, purity, freedom from weeds and diseases, the growers equipment in caring for seed, the general condition of the farm and storage facilities. In cases where these requirements are not met, the field will be rejected for certification. If conditions are found to be satisfactory, the field will be passed and a report of field inspection will be issued to the grower when the inspection is completed.

Laboratory tests

The Inspector of the Crop Improvement Association will take official samples of the seed after it has been processed and sends them for laboratory tests. Processing of seed involves the separation of crop seed from the chaff, broken seeds, inert material, other crop seeds and weed seeds after harvest. This is carried through a mechanical process in which the seed passes through a series of seives and graders. The grower will notify the office of the association when his seed is ready for sampling. Sampling is done in such a manner as to provide samples that are representative of the entire lot. The laboratory tests include the determination of the purity, germination, moisture, colour, plumpness, grade and other factors. (Grading tests are made for seed corn only). When the laboratory tests are over, the association issues the necessary certificates, certification tags and seals to the grower who is now authorized to sell the seed to the cultivators. The tags should contain the following particulars of the seed.

i. Variety.

ii. Season of harvest.

iii. Kind of certification: Whether foundation, registered or certified seed.

iv. Percentage of purity.
v. Percentage of inert matter.
vi. Percentage of other seeds.
vii. Percentage of weed seeds.
viii. Percentage of noxious weed seeds.
ix. Viability.
x. Moisture percentage.
xi. Name of the certified grower.
xii. Date of testing the sample.

There are many reputed and well established large scale seed firms which have branches in more than one State. Most of them have their own experimental stations and put into the market their own bred seed. They have their own seed testing stations and give their own tags for seed produced by them furnishing all the details as required by the law. Most of these reputed firms are not members of the Crop Improvement Association and their seed is not officially certified. However, these private firms have built up such a great reputation among the farmers for their honesty and high quality seed especially on the advent of hybrid corn, that the mere mention of the name of the seed company is itself reckoned as a hallmark for good seed.

There are different certification standards for different crops which specify the minimum requirements. These standards again vary slightly from State to State and may differ from the Federal standards. Usually the State standards are more vigorous and rigid than the Federal standards. For purposes of illustration, the seed standards prescribed for hybrid corn and wheat, oats, barley and rye in the State of Minnesota are given below :—

Commercial Hybrid Corn:

Factor			*Certification Standard*
Pure seed	(Minimum)	..	99%
Inert matter	(Maximum)	..	1%
Weed seed	(Maximum)	..	None
Other varieties	(Maximum)	..	0·5%
Germination	(Minimum)	..	90%
Moisture	(Maximum)	..	13%
Damage	(Maximum)	..	1%
Grading	(Minimum)	..	90%

Wheat, Oat, Barley & Rye

Factor			Standards for each class		
			Foundation	Registered	Certified
Pure seed	(Minimum)	..	99%	99%	99%
Other varieties	(Maximum)	..	0·5%	0·1%	0·2%
Other crop seeds	(Maximum)	..	1 seed/lb.	2 seeds/lb.	5 seeds/lb.
Inert matter	(Maximum)	..	1%	1%	1%
Moisture	(Maximum)	..	13%	13%	13%
Prohibited weed seeds			None	None	None
Restricted weed seeds	(Maximum)	..	2 seeds/lb.	2 seeds/lb.	3 seeds/lb.
Total weed seeds	(Maximum)	..	0·05%	0·05%	0·1%
Germination	(Minimum)	..	No established standard	85%	85%

Seed Storage

Seeds are stored in a variety of ways. Foundation seed is invariably stored in specially built air conditioned store houses which are maintained at low temperatures of about 5°C. and at low relative humidity of not more than 50%. Seed used by breeders for breeding programme and other precious seed is stored in cold storage at—10°C. or lower temperatures. The seed is usually stored in jute bags or some times in paper bags. In this condition the seed retains its viability for several years. Registered and certified seed are stored in large well ventilated damp proof godowns under controlled temperature conditions. The moisture percent of the stored seed is an important factor for consideration. The seed should be thoroughly dried before storage. In regard to oil seeds the moisture content should never exceed 10 prcent, in legumes it may vary from 10 to 12 percent and for cereals the safe limit is from 12 to 14 percent. The seed is stored usually in 100 lb. sacks made of either jute or paper. Many of the private seed companies have huge seed bins mainatined at controlled temperatures. Under these conditions the seed retains its viability for a very long time and remains free of insect attack.

Seed trade in the United States of America

Why American farmers by seed every year? The average American farmer does not keep his own seed as the Indian farmer generally does. This is due to a number of reasons. In the first place, he has no place to store the seed in his house free from insect attack and other damages. Secondly, owing to the mechanisation and use of combines in harvesting, the processing of seed is a major item involving lot of expenditure and effort and is beyond the means of an average farmer. There are large number of seed companies studded throughout the country which have upto-date facilities for seed processing and storage and readily offer pure seed at reasonable prices from whom the seed can be obtained in a few hours' notice at any time in the year. For these reasons the American farmers find it far more easy to buy the seed than to keep their own seed. Further in crops like maize where hyrid seed is sown, it is imperative for the farmer to buy fresh hybrid seed every year as the hybrid crop is unfit for the purpose of seed. Of late, hybrid sorghum has replaced the usual sorghum types and the sorghum growers have to necessarily go in for hybrid sorghum seed every year like the corn

growers. Besides, in a number of vegetables like tomato and onion also the hybrid seed is planted. For these reasons the seed trade in America has been well established.

Seed trade in India

In India, contrary to the well organised seed trade in the United States of America, there is practically no seed trade for field crops. It is the big farmer that keeps the seed and the small farmers get their seed requirements from him at the time of sowing. This is because, it is only the big farmer that has the means and also facilities to store the seed and the small farmer either disposes off all the produce harvested by him or uses it for his bread. Owing to the lack of proper communications from village to village and the shortness of sowing time especially of the rainfed crops, the villagers try to get their seed requirements from within their village itself. They have very little choice of their seed and plant whatever seed is available or given to them by the big farmers or the village merchants at the time of sowing. Even if some farmers want to buy good seed, there is no means for them at present to judge the quality of the seed they are buying. No standards or specifications are prescribed by law to maintain the standards in seed trade. There also seems to be the lack of appreciation of the importance of the good seed by the average farmer; but this is mainly due to want of an opportunity for him to distinguish between good and bad seeds offered for sale. Nevertheless, the Departments of Agriculture, realising the importanoe of good seed, have established a number of seed farms and seed depots for important crops. But the benefit could reach only a fraction of the farming public because of the limited establisment and the vast areas to be covered. Even here, the seed is not certified in the true sense. The value of the seed will increase if a recognised agency certifies the seed and labels each bag giving all the specifications required.

Analysing this situation, it will become evident that the big farmers and the local merchants are the keymen for supplying the seed in our country. Therefore, any scheme for the improvement of the seed must take this factor into consideration. Here is a case, therefore, for organising the Crop Improvement Associations with the co-operation of the big farmers of each village or region. A Crop Improvement Association on the aforesaid lines may be established with the enlightened and big farmers as

its members. They will be required to grow and store seed of the varieties recommended by the Crop Improvement Association. The Departments of Agriculture can effectively co-operate with these associations by lending technical assistance. Suitable seed laws will have to be enacted prescribing the standards for various crops before the Crop Improvement Associations are established. Such a scheme, where the farmers take the responsibility for producing their own seed, is certainly bound to receive the wholehearted co-operation of the cultivators. In our country men of vision and patriotism are not lacking in every village who would come forward to work the scheme successfully.

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Recent Advances in Sorghum Improvement[1]

BY

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SUMMARY

1. Recent advances in the improvement of sorghum are reviewed.
2. The work on interstrain and intervarietal crossing to evolve cosmopolitan types of sorghum are detailed.
3. The problem of the evolution of striga resistant varieties of sorghum is discussed. A method to control striga by adopting a two course rotation of cotton and jonna is suggested.
4. The importance of yellow endosperm in sorghum to increase its nutritive value is indicated.
5. The work on the production of commercial sorghum hybrids by the utilisation of cytoplasmic male sterility is reviewed and the method is described in detail. The future line of work in India for evolving sorghum hybrids is indicated.

Introduction

Sorghum is an important food and fodder crop grown over a very wide area in all warmer parts of the world. Being highly drought resistant it is grown in areas of low rainfall where other crops are less productive. It is believed that this crop has been under cultivation in Africa for several thousand years and has spread to India, China and other Eastern countries. It was introduced into the United States of America only recently where it is now grown over an extensive area. At present sorghum is largely cultivated in India, China, East, West and Central Africa and the United States of America. It is estimated that world's area under sorghum is 116 million acres during the year 1955 and the total production of grain is worked out at 34 million tons of which India contributes 43 million acres with an estimated yield of 6·9 million tons (*Year B. Fd. agric. Statist* 1956).

1. Maharajah of Trivancore, Curzon Endowment Lecture I, delivered at the Agricultural College and Research Institute, Coimbatore, during March 1958 under the auspices of the University of Madras.

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The following are the particulars of area and the total production of sorghum in some of the principal countries where it is grown.

Name of the Country	Area (in million acres)	Production (in million tons)
India ..	42·70	6·93
China ..	19·98	10·85
Central Africa including Egypt ..	3·55	1·78
United States of America ..	2·85	6·06
West Africa ..	1·90	0·36
East Africa ..	0·47	0·12

[Source : Year Book of Food and Agricultural Statistics, U.N.O., 1956.]

Sorghum is considered as a poor man's cereal and some writers, refer to it as a coarse cereal. However studies of the nutritive values of sorghum show that it compares favourably with other cereal grains. According to Colonel Mc Carrison it is richer than rice in protein content, richer than wheat and infinitely richer than rice in fat content (Rangaswamy Ayyangar 1932).

Improvement in sorghum is directed in different ways to suit local requirements. In a country like India where sorghum straws form an important source of fodder, improvement of the stalks besides the grain is also aimed at; while in countries like the United States of America where the straw has practically no economic value, the improvement is directed only towards the grain. Further in this country on the advent of the combines, new varieties of sorghum have been evolved with very short stalks for easy harvest. These combine sorghums are hardly three feet in height while Indian Sorghums are from six to ten feet or more. However special fodder varieties of sorghum are grown in the United States of America which grow to a fairly good height of eight to ten feet. In these fodder varieties the grain has no food value and it is mainly used for seed.

Evolution of Cosmopolitan Strains

In countries where the crop is newly introduced as in the United States of America judicial hybridization and selection have

resulted in the evolution of a number of varieties suited to different rainfall and soil conditions. In countries where the crop has been in cultivation for several thousands of years, as in India, distinct varieties have been formed in different regions. These varieties are at present so highly localised that when grown in a different locality outside their region, they fail to grow so well. Much of the work in sorghum improvement in this country has centered round the evolution of high yielding strains within an existing variety by pure line selection. There being a large number of varieties within comparatively smaller zones and as the varieties in one zone do not thrive well in the adjacent zone, it has become necessary to evolve a large number of strains to suit these different local conditions. For example, in Nandyal Valley of Andhra Pradesh comprising an area of about eight lakh acres, there are three distinct sorghum varieties namely, *Cherukupatcha jonna, Gundupacha jonna* and *Oolapacha jonna.* These three varieties have yellow grain and differ slightly in their panicle shape. The former two varieties have juicy stalks while the latter has pithy stalks. These varieties have become so acclamatised to their respective zones that when they are taken out and sown in the adjacent zone, they do not thrive so well. This is a very great disadvantage for keeping the purity of the strain and also for the multiplication of the seed. Attempts, therefore, have been made at evolving a cosmopolitan variety which will grow equally well in all the three zones of the sorghum growing area by interstrain and intervarietal crossing. Work on these lines has been initiated at the Agricultural Research Station, Nandyal. The preliminary observations in this regard seem to be encouraging.

Control of Striga

Another important problem in sorghum improvement in this country is the control of striga which is a partial root parasite on sorghum. The seeds of striga are very minute and millions of them lie buried in the infected soil. It is estimated that a single striga plant is capable of producing over fifty thousand seeds in one season. When the sorghum seed germinate and the seedlings begin to putforth roots, the striga seed that lie buried in the soil get stimulated into activity. They germinate and attack the sorghum roots and for about five to six weeks the striga remains under ground. Later they push out of the soil rapidly putting forth straight stems and small green lanceolate leaves. They then

flower, set seed and the seed are shed on the soil. The life history is completed in about four to five weeks from the time the striga emerges out of the soil. The parasite does much of its damage even before it shows itself out of the soil. The sorghum crop is stunted and the yields are very greatly diminished. In extreme cases the plants fail to produce earheads resulting in total loss of the crop.

Almost all the varieties in India have been reported to be susceptible to striga. However, a variety from West Africa Bonganhilo was found to be highly resistant to striga during breeding trials at the Millet Breeding Station, Coimbatore (*Madras agric. Stn. Rep.*, 1937-38). Crosses between Bonganhilo and a number of local types were made at the Agricultural Research Stations Nandyal (Kurnool Dist.) and Lam (Guntur Dist.) with a view to evolve striga resistant types suited to local conditions and the performance of their progenies studied. It is observed that these progenies are comparatively more resistant to striga than the susceptible parents. It is further observed that the incidence of striga varies with seasonal conditions. Therefore, the behaviour of the progenies have to be tested through successive generations with reference to their resistance to striga before any definite conclutions are drawn. Another significant observation made in these breeding trials is that the original parent Bonganhilo which is reported to have shown a very high resistance of 97 percent to striga attack at Coimbatore (*Madras agric. Stn. Rep.*, 1937-38) has failed to exhibit the same amount of resistance when raised at Nandyal and Lam where it has shown varying degrees of susceptibility ranging from 13 to 34 percent. Between these two stations also it showed more susceptibility at Lam than at Nandyal. Further it was observed that the attack greatly varied from season to season and year to year. All these observations point to the fact that the resistance to striga is influenced by environmental factors also. Therefore, the evolution of hundred percent striga resistant types through breeding by using the Bonganhilo parent appears to be not very encouraging.

Detailed investigations have revealed that the striga seed is stimulated to germination even by such crops as cotton and cowpea. However these crops do not form the host plants for striga as sorghum and are least affaected by its presence. The striga seed that germinate in the presence of cotton or cowpea soon perish for want of a suitable host. Laboratory experiments have

shown that cotton was found to stimulate the germination of striga seed in much larger numbers than sorghum or cowpea and groundnut had practically no effect (Narasimhamurthi *et al.*, 1957). Here in, therefore, lies a probable solution for the control of the parasite. Sarghum followed by a crop of cotton in rotation will have less incidence of striga than when it follows groundnut or other crops which cannot stimulate striga to germination. This observation is also confirmed by the experience of sorghum growers in the State. Here the cotton crop acts as a check crop in the previous season by stimulating the striga seed to germination which later on perish. It is, therefore, advantageous to adopt a two course rotation of cotton and sorghum in areas where the striga is a bad pest.

Yellow Endosperm

Another important line of improvement of sorghum in recent years is the utilisation of the yellow endosperm character which has been reported to occur in some varieties of sorghums. A variety of sorghum with yellow endosperm was introduced into the United States of America and a number of crosses with this variety and the American types are reported to have been made with a view to introduce this character into American sorghums (*USDA Res. Serv. Spl. Rep.*, 1956). It has also been reported that the carotene content in the yellow endosperm sorghum is nearly equal to that of yellow endosperm maize. In India and other countries where sorghum is an important food crop, the introduction of yellow endosperm into local sorghums will go a long way in improving the nutritional value of the diet of the people of these regions.

Utilization of Hybrid Vigour

The most phenomenal advance in sorghum improvement, made in recent years, is the utilization of hybrid vigour in sorghum. The occurrence of hybrid vigour in sorghum has been reported by many workers. Conner and Karper (1927) and Karper and Quinby (1937) have descrbied the magnitude of hybrid vigour in sorghum. Increase in size of the plant as great as 300 to 400 percent has been observed in several instances. Increase in yields from 10 to 44 percent is also reported. However progress could not be made in the production of hybrid sorghums for commercial seed production, because of the hermaphrodite nature of the spike-

let and the practical difficulties involved in bulk emasculation of the flowers. A method for bulk emasculation of sorghum spikelets was described by Stephens and Quinby (1933). In this method water at 48°C is poured around a sorghum head and left for 10 minutes and this will destroy the viability of pollen. At this temperature the stigmas appear to be less sensitive and are not damaged. The floral parts of some varieties are slightly more sensitive to heat than others and, therefore, the temperature at which the spikelets are to be emasculated will have to be worked out for different varieties and probably for different localities. The hot water method of emasculation of sorghum though relatively simple, requires a supply of warm water in the field and the success depends in preventing water leaking from the container that surrounds the sorghum head. By the very nature of the method it is very cumbersome and difficult to adopt on a large scale for hybrid seed production. At best this method may be useful for a fair supply of hybrid seed for experimental trials.

Rangaswamy Ayyangar and Ponnaiya (1936) and Stephens (1937) discovered a male sterile character in sorghum. In this the anthers were very small with sterile pollen or some times devoid of contents. This character is genetic and is a simple recessive to the normal fertile anthers. Stephens (1937) proposed that this male sterile sorghum might be used for the production of hybrid sorghum seed.

Glen H. Kuykendall (see Quinby and Martin, 1954) discovered a male sterile sorghum plant in a field of the "Day" variety. The "Day" male sterile proved to be a genetic male sterile. When crossed to some varieties, it gave F_1 plants that were sterile or partially so and when crossed to other varieties gave F_1 plants that were fertile. The production of hybrid seed on commercial scale by a three way cross was described by Stephens *et al.* (1952) using this "Day" male sterile character. This method involves the examination of almost every plant in the seed field and the removal of such of the plants which have fertile anthers. Besides losing a good number of plants by this method, there is always the risk of the field getting contaminated by male fertile plants in the seed rows which may fail detection.

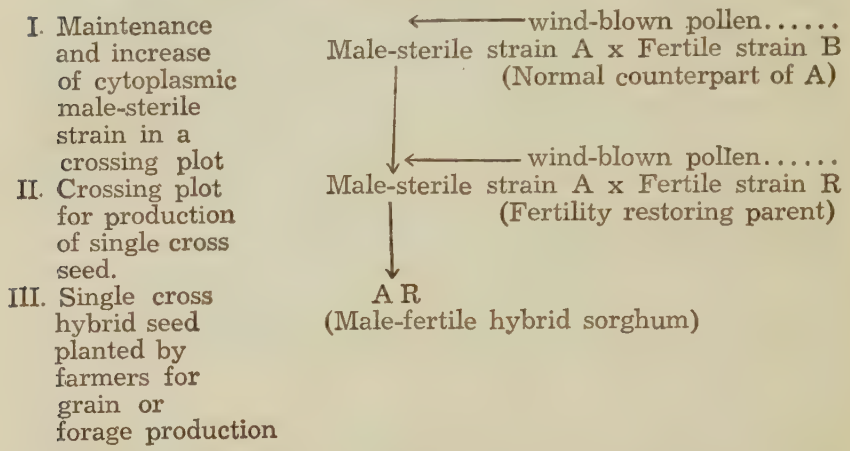
Stephens and Holland (1954) reported the occurrence of cytoplasmic male sterility in sorghum in crosses between sooner Milo and Texas Black-hull Kafir. Partial male sterility was found in F_2 population of these crosses having Milo as female parent.

The degree of sterility increased as the proportion of Kafir chromosomes increased in Milo cytoplasm. By repeated back crossing with Kafir it has been possible to evolve almost cent percent cytoplasmic male sterile types. This cytoplasmic male sterility is genetic and when crossed with certain varieties complete fertility is restored. Each male sterile strain is essentially a female counterpart of the normal strain from which it is principally derived. When pollen from this normal strain fertilises the male sterile strain, the seed from the male sterile strain produces only male sterile plants. Consequently an increased bulk of the seed parent can be grown by planting seed of male sterile plant in the seed rows and the seed of counterpart normal plants in the pollen rows of an isolated field.

When some varieties and strains are used as pollinators on the cytoplasmic male sterile strains they restore complete fertility in the next generation. These are called pollen or fertility restorer strains. When a male sterile strain is used in seed rows and pollen restorer strain in the pollen rows of a crossing block, seed harvested from the seed rows produces fully fertile sorghum hybrids the following year. This is the crop the farmer grows. This type of male sterility has many advantages over the three way cross, for the production of hybrid seed. Since hundred percent sterility is assured in the seed fields the roguing of male fertile plants is eliminated and the purity of hybrid seed is ensured. There is also no loss in the seed fields by the removal of the male fertile plants. The scheme for the commercial production of hybrid seed by using the cytoplasmic male sterile character is briefly outlined below:

Two separate crossing blocks are required in this case. Male sterile strain A is produced in a crossing block in which the pollen parent B is its normal counterpart. A x B will give only cyto plasmic male sterile strain and is identical to A. This crossing percent sterility is assured in the seed fields the roguing of male sterile strain A. In a second crossing block, A forms the seed parent and R is the pollen parent. Strain R is male fertile and is capable of restoring the fertility of A. Thus, A x R gives fertile hybrids. This is the commercial hybrid seed for distribution to the cultivators. The above technique is graphically represented in the following chart.

Method of producing seed of hybrid sorghums.


I. Maintenance and increase of cytoplasmic male-sterile strain in a crossing plot
II. Crossing plot for production of single cross seed.
III. Single cross hybrid seed planted by farmers for grain or forage production
wind-blown pollen.....
Male-sterile strain A x Fertile strain B
(Normal counterpart of A)
wind-blown pollen.....
Male-sterile strain A x Fertile strain R
(Fertility restoring parent)
A R
(Male-fertile hybrid sorghum)

Stephens and Holland (see Quinby & Martin, 1954) observed that cross pollination in sorghum is effective over twelve, forty inch rows. That means under field conditions natural pollination is possible between rows which are situated thirty feet apart. Therefore, it is not necessary to plant the pollinating parent in every alternate row. In actual practice it was found that when two rows of pollen parent alternated with six rows of seed parent (male sterile lines) there was good seed setting. This practice facilitates simultaneous planting of both the parents (either A and B or A and R) by using a four tyned seed drill.

If simultaneous blooming of seed and pollen parents in seed crossing block is a problem, it may be necessary to plant the two parents at different times or to device methods to lengthen the time of pollen shedding. Suitable production practices will have to be developed to suit different situations.

In order to maintain the absolute purity in the crossing blocks, it is necessary to fix the minimum distance between any two crossing blocks, so that even chance cross pollination through wind is avoided. The minimum isolation standard recommended for certified sorghum seed production in the United States of America is a distance of 40 rods which is equivalent to 220 yards. In the case of the production of the breeders' seed, however, the minimum isolation standard is 60 rods or 330 yards. No other sorghum variety should be grown within a radius of this distance from the breeding plots.

A number of hybrid sorghums have been produced in the United States of America and were released to the cultivators for planting. It is reported that in 1955 the Texas Foundation Seed Programme produced 38,000 lb. of sorghum hybid seed (*USDA Res. Serv. Spl. Rep.* 1956). From then onwards the production of hybrid seed was greatly accelerated and at the present day most of the sorghum area in the United States of America is put to hybrid sorghums. The hybrid sorghums have been reported to have given increased yield from 30 to 40 per cent.

The cytoplasmic male sterile character is also used for improvement of forage sorghums. Male sterile seed parents that are both sweet and juicy and that can combine as grain crop are produced. A tall growing sorgo is used as the male parent. The hybrid is vigorous and tall, and is cut for forage. The production of seed in the hybrid is not a factor for consideration since the entire crop is cut and used as fodder and the non-production of seed in the hybrid will be an added advantage since the stalks will then be all the more nutritive.

Trials with some of the American hybrid sorghums at the Agricultural Research Station, Lam and the College Farm, Bapatla were not quite successful. The hybrids were rather short and early. When sown in the main season they were caught in rains and, therefore, have to be grown only in the late season. When compared with the local varieties grown in the late season, the hybrids did not show much promise. The short stalks of the hybrids did not attract the cultivators. Most of the American hybrids have red pericarp which is not very much favoured. Further, the grains have soft endosperm when compared to the horny endosperm of most of the Indian types. The lustre of the seed coat present in the Indian sorghums is lacking in the American hybrids. For these raesons it is very doubtful whether the American sorghum hybrids will make any headway in this country. Therefore, it is very necessary that sorghum hybrids suitable to Indian conditions and taste are evolved by making use of the cytoplasmic male sterile character. A beginning in this direction has already been made at the Agricultural College, Bapatla and a large number of crosses between the American cytoplasmic male sterile lines and Indian sorghum varieties were effected. Examination of the hybrids reveal that some of the hybrids are showing partial male sterility while others are completely fertile. Further crossing and selection work is programmed to evolve suitable cyto-

plasmic male sterile types suited to local conditions. In some instances it is noticed that some of the hybrids between American male sterile types and the Indian restorer varieties are vigorous and tall growing and have a great promise as good fodder hybrids. Further trials with these selections are also in progress. The hybrid sorghums have a great promise in increasing India's food production since sorghum is one of the major food crops in this country.

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